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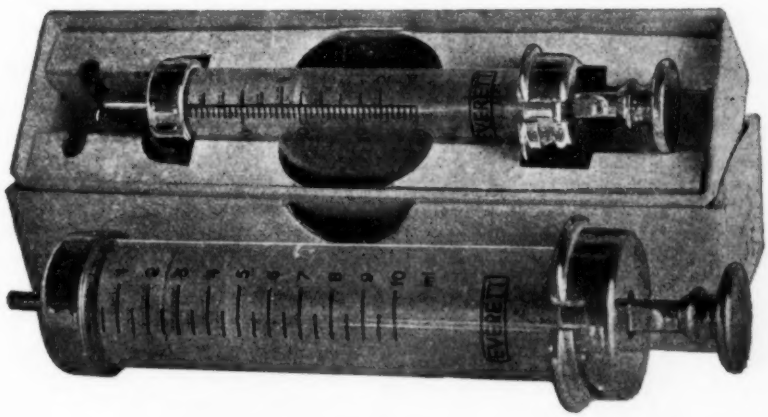

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LOCALIZED TUBERCULOSIS OF THE DESCENDING COLON

L. STEIN, B. SC.HONS., M.B., B.CH. (Rand), F.R.C.S. (EDIN.)
and
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Compared with the small intestine, the colon is rarely the site of tuberculosis and when infection occurs it is almost always near the ileo-caecal region.[1] McLaughlin[2] states that 85% of intestinal tuberculosis occurs in the ileo-caecal region and the ascending colon, probably because lymphoid patches are numerous there or because of the relative stagnation of contents in the lower ileum. Widespread ulceration may affect a large part of the large intestine as a terminal event in a patient suffering from active pulmonary tuberculosis.[3] In a series of 5,529 admissions of cases of pulmonary tuberculosis during the 25 years 1924-49, Mitchell and Bristol[4] found the intestine involved in 24·7% of advanced cases.

Apart from the rectum and anus, which will not be discussed, localized tuberculous disease of the large bowel distal to the caecum and ascending colon is uncommon. Boles and Cohen[5] in a series of 1,000 consecutive autopsies found 23% with healed or active tubercle, of which 28% (63 cases) showed intestinal tuberculosis. There were no cases of localized colonic tuberculosis beyond the ascending colon and there have been very few such cases reported. Three involving the hepatic flexure,[6] and 3 the transverse colon,[7-9] the last two of which were associated simultaneously with carcinoma, have been described. The double lesions of carcinoma and tuberculosis have been noted in the sigmoid colon, [10-12] and Miller[13] has described a case in which the patient died of phthisis and at post-mortem a mass was found in the descending colon which was mixed tubercle and carcinoma on section. No reports of localized tuberculosis of the descending colon, diagnosed during life, could be found in the literature. At Baragwanath Hospital during the 5½-year period, January 1951 to June 1956, 284 cases of intestinal and peritoneal tuberculosis were diagnosed, and of these only one case affected the colon distal to the ascending colon. This was of a tuberculous lesion localized to the descending colon and it was felt that it was rare enough to warrant this report.

CASE REPORT

J.B., a 50-year-old Bantu male was admitted to Baragwanath Hospital on 15 May 1956 complaining of pain in the right axilla and in the left side of the abdomen, both of 2 weeks' duration. The abdominal pain was colicky in nature and did not radiate. There was no vomiting and no abnormality in the stools or bowel action.

Examination

The patient looked rather ill, but was apyrexial. In the right axilla was a large tender mass which consisted of a matted fixed group of glands associated with what appeared to be a typical discharging tuberculous sinus. In the left groin and continuing to the left iliac fossa was a fairly tender ill-defined mass, 3 inches by 1 inch in size, which was firm and appeared to be fixed to the posterior abdominal wall. No enlargement of liver or spleen was felt.

Investigations

The stools were negative for amoeba and several urine examinations for tubercle appeared negative. Biopsy of the axillary gland showed a tuberculous adenitis. X-ray of the chest showed a slight pleural reaction at the left base, but the lungs were otherwise clear. Retrograde pyelogram was normal. Barium-enema examinations on 24 May and 1 June revealed a 3-inch regular smooth narrowing in the descending colon, which halted the flow of barium temporarily (Fig. 1). The mucosa in the narrowed segment was intact (Fig. 2). The remainder of the colon and the caecum and terminal ileum appeared normal.

It was felt that the stricture was most likely tuberculous in origin in view of the presence of the tuberculous axillary gland and the intact mucosa.

The patient was put on an anti-tuberculous regime of strepto-

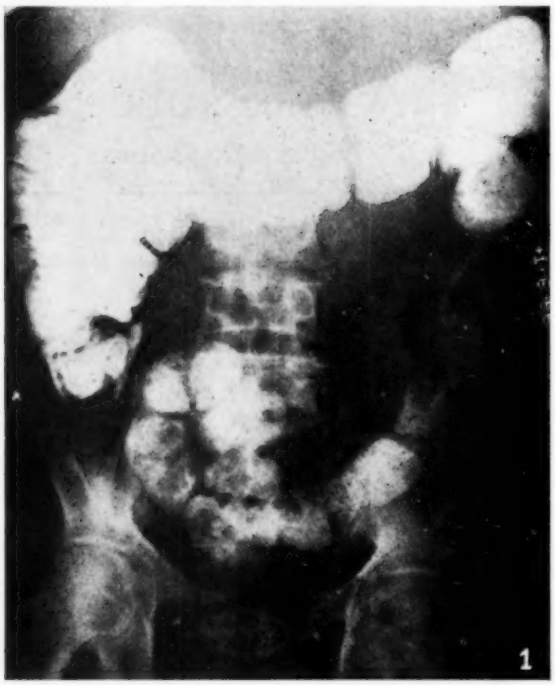

Fig. 1. Barium enema to show the narrowed area in the descending colon.

mycin, Rimifon and PAS daily for 6 weeks. At this stage he developed symptoms and signs of partial intestinal obstruction and the mass now felt stony hard, non-tender and fixed. A repeat barium-enema examination showed exactly the same appearances as the first. On 30 June laparotomy was performed in view of the patient's age, the possibility of the mass being carcinomatous, and the possibility of increasing obstruction.

Operation Findings

The mass was found to be due to a very thickened descending colon surrounded by numerous adhesions. The posterior peritoneum was also markedly thickened. The greater omentum was found to be shrivelled up and thickened to form an 'omental cake'. The caecum and remainder of the colon appeared normal. After the descending colon and splenic flexure were with some difficulty mobilized, they were resected and an end-to-end anastamosis of the terminal portion of the transverse colon to the sigmoid colon was performed. The resected portion on being opened was found to present the typical appearances of hyperplastic tuberculosis, viz. markedly thickened wall with extensive fibrous-tissue infiltration giving rise to a very narrow lumen.

Histological Report by Dr. I. W. Simson of the South African Institute for Medical Research:

'Section from the specimen of descending colon shows the presence of numerous tubercle follicles lying in the submucosa. The serosal surface is markedly thickened owing to fibrous and granulation tissue in which there are areas of coagulative necrosis surrounded by endothelioid cells and histiocytes. In addition, an occasional acid-fast bacillus was also observed. The histological features are those of tuberculosis.'

Progress

The patient made a good recovery. He was seen 3 months after the operation and was fit and well, having gained a little weight. There was no disturbance of bowel function. Anti-tuberculous treatment was continued for 6 weeks after the operation.

DISCUSSION

Carcinoma is the common lesion causing a stricture in the descending colon. Characteristically the stricture is irregular, fixed, with the loss of mucosa, and often eccentric. It is sharply defined and constant, which distinguishes it from colospasm.[14] In our patient the stricture was unchanged in appearance after 6 weeks and, the mucosa being intact, a non-maliganant condition was suggested. However, a disturbed mucosal pattern does not exclude tubercle as Eckey's case showed altered mucosa and was erroneously diagnosed as neoplasm.[15]

The alteration in the consistency of the mass after treatment has been commented on by Bohm,[16] who stresses that a tuberculous hyperplastic mass, after anti-tuberculous treatment, may be indistinguishable from carcinoma of the colon. This therapy was also probably responsible for the absence of fistula formation after the operation. This complication was common before the advent of the modern anti-tuberculous drugs, and Kallqvist[17] maintains that the drugs have a prophylactic action against secondary intestinal tuberculosis. In spite of this, however, it is surprising that in our patient acid-fast bacilli were still found in the tuberculous mass and also that there was no change in the barium-

Fig. 2. View showing that the mucosa in the narrowed area is intact.

enema appearances, even after intensive treatment lasting 6 weeks.

SUMMARY

A case of localized tuberculous lesion of the descending colon is described. This condition has been very rarely reported.

We should like to thank Dr. H. Clain for allowing us access to the radiological findings, and to the Superintendent of Baragwanath Hospital for permission to publish this case. We should also like to thank Dr. M. Schwartz for referring the case and Mr. Shewitz of the Department of Medicine for help with the photographs.

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ASSOCIATION NEWS : VERENIGINGSNUUS

UROLOGICAL CONGRESS AT PORT ELIZABETH

History was made on 5, 6 and 7 November 1956 at Port Elizabeth When the Urological Association of South Africa held their first independent Group Congress. The following 15 members attended, viz. Messrs. H. C. Brayshaw, C. D. Kisner, E. Lipworth and R. D. Venn, from Johannesburg; Mr. S. McMahon from Durban; Messrs. A. J. S. Burger, J. A. Currie, P. J. M. Retief and R. B. Watson, from Cape Town; Messrs. L. Jordaan and I. P. Maas, from Pretoria; Mr. A. J. Vorster from Bloemfontein; Mr. R. McK. Honey from Salisbury and Messrs. W. F. de Villiers and J. C. Jordaan, from Port Elizabeth.

The proceedings commenced on Monday 5 November with an address by the President, Mr. S. McMahon, on 'Surgery of the Ureter'. This was followed by a business meeting and the election of office-bearers for the ensuing year; viz. *President* Mr. Brayshaw, *Hon. Secretary* Mr. Retief and *Committee Members*, Messrs. J. A. Currie, J. D. Joubert, S. S. Scher and R. B. Watson. It was unanimously agreed to take time off during the afternoon to watch the MCC *vs* EP cricket match, and in the evening a paper *Akute Renale Ineenstorting* was read by Mr. Leon Jordaan from Pretoria.

Tuesday morning 6 November was devoted to a symposium on the Neurogenic Bladder, when the following papers were read:

'Anatomy and Physiology of the Bladder'—Mr. Retief.
'Emergency Treatment of Neurogenic Bladder—Mr. Currie.
'Assessment of Neurogenic Bladder'—Mr. Kisner.
'Surgical Treatment of Neurogenic Bladder'—Mr. Brayshaw.

On Tuesday afternoon there followed a presentation of short papers and clinical cases. Mr. Lipworth discussed his experience in surgical repair of Hypospadias, and Mr. McMahon recounted his treatment of a rare case of Leiomyosarcoma of the Bladder. This was followed by clinical cases with X-rays:

Mr. Brayshaw: 'Several cases with ectopic ureters', and 'Extravasation occuring with urethrograms'.

Mr. Retief: 'Prostatic obstruction with gross renal regurgitation', and 'Papillary carcinoma of the kidney'.

Mr. Currie: 'Hytadid of the kidney'.

Mr. Lipworth: 'Ureteral calculus removed by rectal manipulation'.

Mr. Honey: 'Brucellosis of the kidney'.

On Wednesday 7 November all the members attended a ward round at the Livingstone Hospital conducted by Mr. James Jordaan, which proved most interesting and provoked much discussion.

The interest shown in this the first Congress of the Urological Group made all members hopeful for its continuance in the years between the major Congresses of the Medical Association. The members also decided to arrange for a symposium by the Urological Section on Surgery of the Urethra at the next South African Medical Congress at Durban in 1957.

A farewell luncheon was held at the P.E. Club, at which Mr. Brayshaw thanked Messrs. de Villiers and Jordaan for their extensive and excellent hospitality and for the very fine way in which all the arrangements for the Congress had been made. It should also be recorded that Mr. Mass, the outgoing Secretary, had enormously influenced the success of the gathering by his continued enthusiasm throughout the year.

BOOKS RECEIVED : BOEKE ONTVANG

Der Rheumatismus. By Prof. Dr. R. Hopmann. Pp. 100. 29 Illustrations. DM 16.50. Stuttgart: Georg Thieme Verlag. 1956.

EKG-Fibel. By Dr. R. Heinecker. With a Preface by Prof. Dr. F. Hoff. Pp. xii + 200. 182 Illustrations in 308 Single Representations and 34 Schematic Representations. DM 19.80. Stuttgart: Georg Thieme Verlag. 1956.

Die Lungentuberkulose. Diagnose und Therapie. Third Edition. By Prof. Dr. P.-G. Schmidt. Pp. xii + 384. (Some in colour.) DM 58. Stuttgart: Georg Thieme Verlag. 1956.

The Queen Charlotte's Textbook of Obstetrics. Ninth Edition. By Members of the Clinical Staff of The Queen Charlotte's Maternity Hospital, London. Pp. viii + 547. 4 Coloured Plates and 246 Text Figures. 45s. net. London: J. & A. Churchill Ltd. 1956.

Ciba Foundation Symposium on Bone Structure and Metabolism. Editors for the Ciba Foundation. G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch. and Cecilia M. O'Connor, B.Sc. Pp. xii + 299. 121 Illustrations. 45s. net. London: J. & A. Churchill Ltd. 1956.

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South African Medical Journal

EDITORIAL

PERCUTANEOUS ABSORPTION

The application of ointments to the skin for cosmetic and medicinal purposes goes back to antiquity, but this practice has until recent years been on an empirical basis. Modern advances in dermatology have given a better understanding of the efficacy of such preparations,[1] and emphasis has been placed on the use of different ointment bases, the nature of which may well determine the usefulness of a particular ointment as much as the active ingredient. Many different synthetic bases such as the polyethylene glycols are now available in addition to the traditional fatty materials of animal or mineral origin.

Few drugs can readily penetrate the intact skin and as a method of administration of drugs the percutaneous route has limited value. Nevertheless it may prove useful in certain cases; for example a high concentration of drug can be maintained for intense local action in a particular part of the body, and it may prove effective as an alternative method of administration where oral or parenteral therapy is not feasible. Very important is the recognition of the hazards from percutaneous absorption of many toxic agents used in industry and agriculture.

Several factors determine the degree of absorption of a medicament from the skin. First of all it must penetrate the skin, the main route being through the hair follicles and the sebaceous glands, although some penetration may also occur through the sweat glands. After the drug has passed down the hair follicle the conditions are favourable for absorption. An increase in the skin temperature enhances the absorption of the inuncted material, presumably by lowering the viscosity of the sebum and by the increase in the blood supply. The solubility of the drug in lipoids is important, or its ability to combine with fatty acids in the sebum; the metallic ions of ammoniated mercury may ultimately be absorbed by such a mechanism. The organic phosphorus insecticides such as parathion are soluble in lipoids and water so that absorption of these dangerous agents occurs readily. An ointment base may retard penetration and absorption, or it may

VAN DIE REDAKSIE

PERKUTAANSE ABSORBERING

Die aanwending van salf aan die vel vir kosmetiese en geneeskundige doeleindes dateer terug tot die Oudheid, maar hierdie gewoonte was tot 'n paar jaar gelede nog op 'n proefondervindelike basis. Moderne vooruitgang in huidsiekteleer het 'n beter begrip van die werksaamheid van sulke preparate[1] gegee, en klem is gelê op die gebruik van verskillende salfbasisse, die aard waarvan net sowel die bruikbaarheid van 'n besondere salf as die aktiewe bestanddeel mag bepaal. Behalwe die tradisionele vetstowwe van dierlike- of minerale-oorsprong, is baie verskillende sintetiese basisse, soos byvoorbeeld die poli-etileen glikole, nou beskikbaar.

Min geneesmiddels kan maklik deur die ongeskonde vel dring, en as 'n metode van toediening van geneesmiddels, het die perkutaanse roete 'n beperkte waarde. Nogtans mag dit by sekere gevalle nuttig blyk; 'n hoë konsentrasie van geneesmiddel kan byvoorbeeld vir intense lokale werking in 'n besondere deel van die liggaam volgehou word, en dit mag effektief blyk as 'n alternatiewe toedieningsmetode waar mondelinge of parenterale terapie nie doenlik is nie. Dit is baie belangrik dat die gevare deur perkutaanse absorbering van baie giftige stowwe wat in industrie en landbou gebruik word, besef word.

Verskeie faktore bepaal die mate van absorbering van 'n geneesmiddel deur die vel. Eerstens moet dit deur die vel dring, aangesien die hoofroete deur die haarsakkies en die smeerkliere is, alhoewel daar ook 'n mate van deurdringing deur die sweetkliere mag geskied. Ná die geneesmiddel langs die haarsakkie afgegaan het, is die toestande gunstig vir absorbering. 'n Verhoging in die temperatuur van die vel verhaas die absorbering van die ingevryfde stof, vermoedelik deur die viskositeit van die huidsmeer te verlaag, en deur die verhoging in die bloedtoevoer. Die oplosbaarheid van die geneesmiddel in lipoïede is belangrik, asook sy vermoë om met vetsure in die huidsmeer te kombineer; die metaalagtige ione van ammoniakkwik mag uiteindelik deur so 'n meganisme geabsorbeer word. Die insektemiddels met organiese fosfor, soos byvoorbeeld paration, is oplosbaar in lipoïede en water, met die gevolg dat absorbering van hierdie gevaarlike stowwe maklik geskied. 'n Salfbasis mag deurdringing en absorbering vertraag, of dit mag die oordrag van 'n geneesmiddel, weens sy mengbaarheid met die huidsmeer, vergemaklik. In hierdie laasgenoemde verband het dit

facilitate transfer of a drug by its miscibility with the sebum. In this latter respect animal and vegetable oils and fats have proved superior to mineral fats.

Various modern synthetic agents have been introduced to enhance the percutaneous absorption of drugs, but more research is needed to find the best compounds for this purpose. In assessing their value many factors need to be taken into account in addition to those considered above. Thus the method of application, which may include friction, massage, and mechanical pressure, and the duration of application, must be taken into consideration. The methods that have been used measure either skin penetration or systemic absorption. Comparisons need to be made with standard preparations under the same conditions. At the best it is usually only possible to make general statements, since strictly valid mathematical assessments can rarely be made. Microscopic sections or auto-radiographs or chemical methods have been used to determine penetration of the skin by specially selected agents. For the assessment of absorption physical and chemical tests have been performed on blood, urine, faeces, or a particular tissue, while radio-active substances have increased the value of such methods. Pharmacological effects have also been measured in studies of the absorption of certain drugs.

In recent years certain oil-soluble vitamins and hormones have been administered through the skin. In this way vitamin-K preparations have been used in newborn infants. As far as oestrogens are concerned the natural hormones are found to be better absorbed than synthetic compounds, for instance to promote breast development in the hypogonadal female; their use in cosmetic preparations may produce systemic effects and is possibly not without danger.

Apart from the sensitization that may occur when certain drugs are applied to the skin, there is the danger of harmful systemic effects when chemicals are absorbed from the skin, especially if large areas of damaged skin are the site of application. Even such apparently innocent substances as boric acid and borax have caused fatal poisoning in babies in this way.

1. Hadgraft, J. W. and Somers, G. F. (1956): J. Pharm. Pharmacol., **8**, 625.

geblyk dat dierlike en plantaardige olies en vette baie voortrefliker as minerale vette is.

Verskeie moderne sintetiese stowwe is ingestel om die perkutaanse absorbering van geneesmiddels te verhaas, maar verdere navorsing is nodig om die beste verbindings vir hierdie doel te vind. By die bepaling van hulle waarde moet baie faktore, behalwe dié wat hierbo oorweeg is, in ag geneem word. Dus moet die metode van aanwending, wat vrywing, massering en meganiese druk kan insluit, asook die duur van die aanwending, in ag geneem word. Die metodes wat gebruik is, meet òf deurdringing van die vel òf absorbering in die liggaam. Vergelykings behoort gemaak te word met standaard preparate onder dieselfde toestande. Op die beste is dit gewoonlik slegs moontlik om algemene bewerings te maak, aangesien streng matematiese berekenings selde gemaak kan word. Mikroskopiese deursnitte, of outoradiograwe, of chemiese metodes is gebruik om deurdringing van die vel deur spesiaal uitgesoekte stowwe, vas te stel. Vir die berekening van absorbering is fisiese en chemiese toetse op bloed, urien, ontlasting, of 'n besondere weefsel uitgevoer, terwyl radio-aktiewe stowwe die waarde van sulke metodes laat toeneem het. Farmakologiese uitwerkings is ook by hierdie studies van die absorbering van sekere geneesmiddels gemeet.

Gedurende die afgelope jare is sekere vitamiene en hormone wat in vet oplosbaar is, deur die vel toegedien. Preparate van vitamien-K is op hierdie wyse by pasgebore suigelinge gebruik. Wat estrogene betref, is dit gevind dat natuurlike hormone beter geabsorbeer word as sinteitese verbindings, soos byvoorbeeld om borsontwikkeling by 'n hipogonadale vrou te bevorder; hulle gebruik in kosmetiese preparate mag uitwerkings op die liggaam veroorsaak, en is waarskynlik nie sonder gevaar nie.

Behalwe die gevoeligmaking wat mag voorkom wanneer sekere geneesmiddels aan die vel aangewend word, is daar die gevaar van skadelike uitwerkings op die liggaam as chemikalieë deur die vel geabsorbeer word, veral waar die gebied van aanwending groot gedeeltes van beskadigde vel is. Self sulke skynbaar onskuldige bestanddele soos boorsuur en boraks het op hierdie wyse noodlottige vergiftiging by babas veroorsaak.

1. Hadgraft, J. W. en Somers, G. F. (1956): J. Pharm. Pharmacol., **8**, 625.

INFESTATION BY FASCIOLA HEPATICA

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Although *Fasciola hepatica* (liver fluke, *Lewerbot*) is essentially a parasite of herbivorous animals, especially sheep and cattle, in which it causes the disease known as 'liver rot', it has been reported in man as an erratic infestation. The recent recognition of 2 human cases, one presenting with massive haemorrhage from the liver into the intestine, and the other with intermittent obstruction of the common bile-duct, prompted an extensive review of the literature, from which it became apparent that fascioliasis is not so very rare in man, in whom it may produce variable clinical pictures.

It is the purpose of this paper to draw attention to the

pathological effects of infestation by *F. hepatica*, to discuss the clinical features, diagnosis and treatment of fascioliasis (distomiasis), and to report the 2 cases encountered.

Fasciola hepatica (*Distoma hepaticum*), a flat, leaf-shaped worm, pale-grey in colour with dark borders (Fig. 1), belongs to the class Trematoda or flukes. It is related to *Chlonorchis sinensis*, the common liver-fluke of Eastern races, and also to the blood flukes (Schistosoma

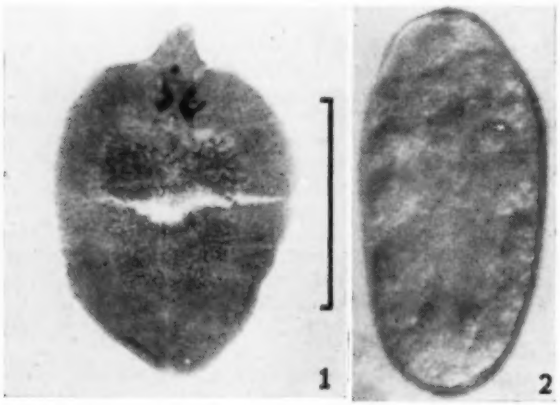

Fig. 1. *Fasciola hepatica.* Adult fluke (the scale represents 1 cm.).

Fig. 2. The ovum of *F. hepatica.* Note the relatively small operculum.

or Bilharzia), common in Africa. The adult is 20-30 mm. long, and 10-15 mm. broad. The conical head-end is surmounted by an oral sucker, behind which is a larger, triangular ventral sucker. The eggs (Fig. 2) measure 130-180 microns by 70-90 microns. They are ovoid, have a yellowish-brown operculated covering or shell, and contain the ovum and yolk-sac.

LIFE CYCLE

The parasite requires two hosts for reproduction (see Fig. 3): The *definitive host*, in which the adult fluke develops, is found chiefly in domestic animals, especially sheep, cattle and goats, and rarely horses and pigs. The fluke also occurs in the other plant-eating animals, e.g. buffalo, hare, rabbit, elephant, hyrax (dassie) and kangaroo, and occasionally in domestic carnivora, e.g. dog and cat.

The *intermediate* hosts are fresh-water snails of the genera Limnaea and Physopsis. In Europe the species is *L. truncatula* in Japan, *L. pervia*; in South Africa, *L. natalensis*, *Physopsis africana* and *Bulinus verreauxii*.

The adult fluke lives in the bile-ducts of the definitive host. Ova are discharged into the bile, and pass down the intestinal canal to be excreted in the faeces. The eggs may thus reach fresh-water ponds, where they can remain viable for 2-3 weeks. The ciliated embryo or miracidium which hatches from the egg cannot survive for more than 8 hours in the water. However, if it finds a snail the life cycle is continued. In the snail the miracidium

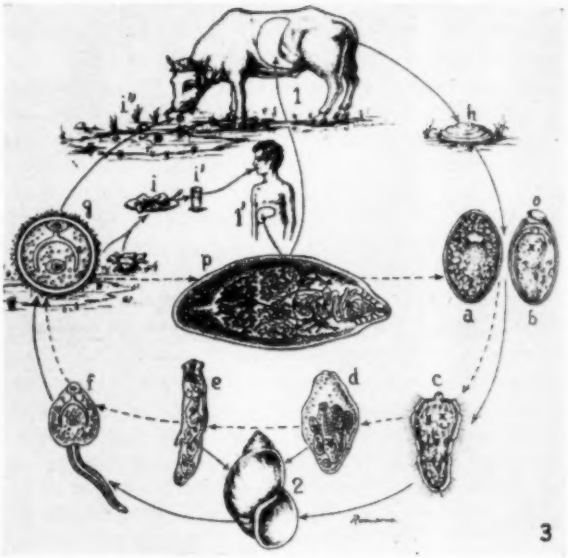

Fig. 3. *Fasciola hepatica.* Life cycle. The adult parasite (p) lives in the bile ducts of the definitive host (cattle, 1; man, 1′). The eggs (a) are passed in the faeces (h). After reaching the water they become embryonated (b), the miracidium (c) hatches through the operculum (o), and swims freely in the water until finding the intermediate host (2) (*Lymnea cubensis* and *Physa cubensis* in Cuba), where the sporocysts (d), rediae (e) and cercariae (f) develop. The cercaria (f) leaves the snail and becomes encysted in the water either freely or attached to water plants, developing into the metacercaria (g), which is the infective stage for the definitive host (1, 1′). When the definitive host ingests the metacercaria with water or vegetables (i, i′, i″) it reaches the intestine, the cystic cover dissolves and frees a young fluke; this bores through the intestinal wall and reaches the liver surface through the peritoneum; when boring through the fibrous capsule and the liver parenchyma, the parasite produces necrosis; in the bile it becomes an adult hermaphroditic parasite and lays eggs which are passed in the faeces (h), beginning a new cycle. (*After Drs. Kouri and Basnuevo.*)

develops into a sporocyst which liberates up to 20 rediae. Each of these produces about 20 cercariae which escape from the snail and swim about freely in the water.

After about 8 hours the cercariae lose their tails, and encyst on blades of grass or aquatic plants (watercress), which are eaten by the mammalian hosts. The encysted cercariae may survive for long periods, e.g. several weeks in dry hay, the whole winter in water, and for a year or more in damp hay.

After the cercaria has been ingested by the definitive host, its outer covering is dissolved in the alimentary canal of the animal, and a young fluke emerges. These young flukes are attracted to the host's liver by positive chemotaxis, and they reach it by a variety of routes. Most commonly the flukes burrow through the intestinal wall and migrate transperitoneally (compare the similar migration by *Paragonimus*). Less commonly they reach the liver via the portal blood-stream or lymphatics (compare the migration of Schistosoma) or very rarely they pass up the common bile-duct (compare the passage

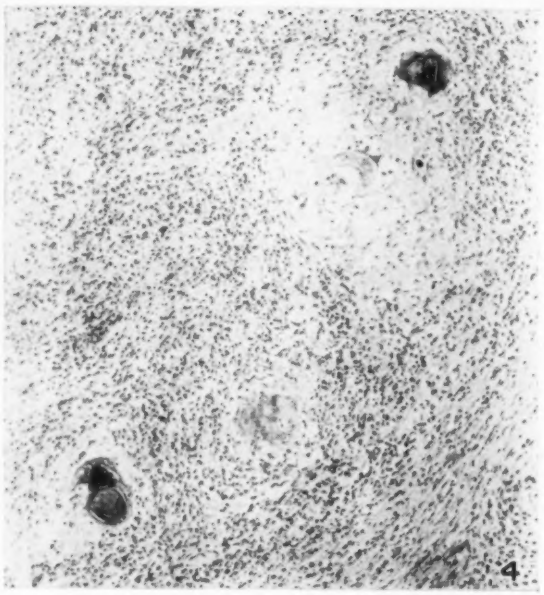

Fig. 4. Microphotograph showing distorted ova of *F. hepatica* surrounded by localized foci of pseudo-tubercles in the connective tissue surrounding the cystic duct.

of *C. sinensis* and *Ascaris*.) Occasionally the flukes are deflected from the liver and settle in neighbouring tissues, e.g. abdominal wall, spleen, stomach, appendix, etc., or they may pass through the liver to reach the systemic circulation and lodge in distant organs, e.g. cerebrum, kidney, peripheral veins, or even the foetus of pregnant animals.

The transperitoneal migration of the young flukes may be questioned, but the following observations appear to support this method of spread:

1. Small haemorrhagic foci are often found on the peritoneal surfaces of the intestine and liver along the path of migration of the flukes.

2. Ectopic foci of infestation are usually in the neighbourhood of the liver, particularly in the subcutaneous tissues of the right hypochondrium.

3. In our first case ova were found in the connective tissue *outside* the cystic duct and not in the lumen or wall of the duct (Fig. 4).

The young flukes wander about in the liver parenchyma until maturity is reached in 6-12 weeks. The adult eventually reaches the larger bile-ducts where it feeds on blood, liver tissue and bile. In animals the parasite usually survives for 9 months, but it may live for 5 years and longer. It is possible that *F. hepatica* may exist in man for even longer periods (*C. sinensis* has a life-span of 12 years, and schistosomes may survive for 30 years.)

DISTRIBUTION

Infestation of animals has been reported from all parts of the world, but particularly from sheep- and cattle-raising countries with marshy areas. The disease is particularly common in the West Indies, Central America and Central Africa. In the Far East it is much less common than clonorchiasis. In some parts of the British Isles 20-25% of sheep and cattle are infested, 90% of them coming from marshlands.

In South Africa animal infestation is fairly general in the summer-rainfall areas. Up to 75% of sheep and cattle have been infested with *F. hepatica* and *F. gigantica* in certain parts of the Bethlehem and Utrecht districts of the Free State, in portions of the Carolina, Volksrust and Ermelo districts of the Transvaal, and in various inland areas of Natal. In the winter-rainfall areas (Cape Western Province) infestation is much less common, and only occasional cases are seen, although isolated epidemics localized to particular farms have been reported. In the dry areas such as Namaqualand and South West Africa, infestation is very rare. The percentage infestation at various abattoirs in the Union is given in Table I. This naturally depends on the areas from which slaughter animals are drawn. Thus Pretoria, which draws its animals mainly from the Eastern Transvaal, where infestation is common, has a high incidence, while Johannesburg, which draws animals from all parts of the Union, has a comparitively low incidence.

TABLE I. PERCENTAGE INFESTATION AT MUNICIPAL ABATTOIRS

	Sheep	*Cattle*
Cape Town ..	0·06%	0·4%
Port Elizabeth ..	0·28%	3·42%
East London ..	3·5—5%	8·7%
Durban	2·7%	7·6%
Pietermaritzburg ..	'Seen almost daily'	'Seen almost daily'
Pretoria	10% *F. hepatica* and *F. gigantica*	10% *F. hepatica* and *F. gigantica*
Johannesburg ..	0·23%	1·47%
Kimberley	Nil	0·02%
Windhoek	Cattle and Sheep 0·47%	

Cases of human infestation have been noted in Cuba, Venezuela, Argentine, France, Germany, Hungary, Salonika, the Dardanelles, Belgian Congo, Moçambique, and the Far East. No South African cases have hitherto been reported. Human infestation appears to be particularly common in Cuba, whence two large epidemics have recently been reported. In the epidemic of 1944 a single doctor attended to over 40 affected patients, all of whom had eaten watercress. In the 1947 epidemic, which occurred in the same province, 52 patients were seriously affected. Apart from these there have been sporadic outbreaks where several members of a family were affected.

PATHOLOGICAL EFFECTS

When the definitive host is first infected the parasite or its waste products may be responsible for anaphylactic reactions (compare the swimmers' itch of schistosomiasis, and reactions to hydatid disease and ascariasis).

The major effects are due to destruction of the liver and irritation of the bile-ducts by the flukes. The young flukes destroy the hepatic parenchyma as they move about, leaving haemorrhagic tracts in their wake. As they grow the amount of destruction increases, resulting in multiple areas of focal necrosis and considerable loss

of blood with consequent anaemia. Heavy infestation, which is common in sheep but rare in man, causes great enlargement and congestion of the liver, with numerous small dark-red areas of necrosis containing the young flukes. Blood and parasites pass into the larger bile-ducts, and then into the intestines. Massive infestation may result in rupture of the liver capsule, with intra-peritoneal haemorrhage or frank bleeding into the intestine *via* the bile-ducts. This is the usual cause of the sudden deaths which occasionally occur in animals and, although it has not been reported in man before, was a major feature of our first case. In most of the human cases that have come to operation or autopsy the liver has been enlarged, with multiple small 'abscesses' containing necrotic material and a well-marked peri-hepatitis. This was well demonstrated in our first case, where the liver was studded with numerous necrotic abscesses, the largest of which were about 5 cm. in diameter. These were lined by vascular granulation-tissue, surrounded by epithelioid cells and giant cells and a peripheral zone with eosinophilic infiltration. (Others have noted the presence of Charcot-Leyden crystals in this zone.) In both animals and man these necrotic areas are liable to secondary infection. In sheep infection by *Cl. oedematiens* causes the condition referred to in Australia as 'black death'. In man pyogenic infection results in a septic cholangitis (Case 1).

As the disease becomes more chronic the effects of irritation of the mucosa of the bile-ducts by the adult flukes become manifest. A chronic cholangitis is produced (Fig. 5), with gross thickening (up to ½ cm.) of the walls of the ducts, which become visible through the capsule of the liver as small yellowish streaks (in man) or thick white cords (particularly in cattle—Fig. 6).

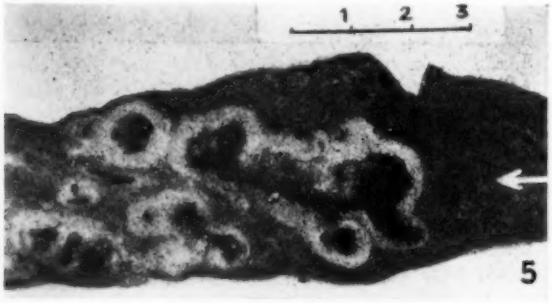

Fig. 5. Photograph of cut surface of ox liver showing grossly thickened and dilated hepatic ducts with calcified walls. The arrow points to an adult fluke. The scale shows 3 cm.

These cords are best seen on the inferior surface of the left lobe of the liver, which is usually mostly affected. In long-standing infestations the ducts become converted into inert, convuluted tubes, expanding in places into cavities or diverticula as large as a hen's egg, and lined by bile-stained fibrous tissue and calcareous material (Fig. 5). They contain numerous parasites and ova, epithelial debris, sandy concretions and muco-pus. The epithelial lining becomes markedly hyperplastic, and may undergo malignant changes (compare bilharziasis).

Fig. 6. Photograph of the under-surface of ox liver. The grossly thickened bile-ducts are visible through the capsule of the liver.

Anaemia is a common accompaniment, and is due mainly to loss of blood from the liver and to a lesser extent to a haemolytic toxin produced by the parasite or bleeding from oesophageal varices.

The adult flukes also tend to irritate and block the extra-hepatic bile-ducts, and give rise to jaundice (case 2). A striking feature is an intense inflammatory reaction in the walls of the ducts and peri-ductal connective tissue, which was well illustrated in our second case. Biliary stasis and the presence of foreign material in the ducts predispose to the formation of primary common-duct stones in both animals and man, and also to ascending septic cholangitis.

The gall-bladder is often normal although distended (both our cases), but it may show evidence of chronic inflammation with or without stones, or even an empyema. It is exceptional to find flukes in the gall-bladder (only 1 human case reported). Our first case was of interest in that there was an intense inflammatory reaction in the connective tissue surrounding the cystic duct, with diffuse infiltration of plasma cells, macrophages and eosinophiles and, in addition, localized foci of pseudo-tubercles surrounding recognizable ova (Fig. 4).

From the above it should be clear that *jaundice* may be due to a variety of causes, viz. multiple intra-hepatic 'abscesses' with or without cirrhosis, septic cholangitis, and obstruction of the common bile-duct by adult flukes (case 2), gall stones, or even blood clot (case 1). However, jaundice is not a constant feature and does not necessarily accompany hepatomegaly.

In ectopic sites, e.g. subcutaneous tissues, appendix, spleen, brain etc., similar granulomatous 'abscesses' are

produced. In addition, multiple haemorrhagic foci have been described in the lungs and a fibrinous peritonitis may occur expecially in cattle. Catarrhal changes in the intestinal mucosa are common, and may be due to irritation by the flukes or their waste products.

In the late stages of the disease there is gross wasting and oedema with hydrothorax and ascites. Terminal pulmonary lesions occur in animals and man.

SYMPTOMATOLOGY IN ANIMALS

In rare *acute cases* the animal dies suddenly with blood-stained froth appearing at the nostrils and the passage of blood *per anum*.

In *ordinary cases* the disease is insidious in onset, and the first symptoms occur when the young flukes have reached a fair size.

Sheep. At first the sheep shows a tendency to fatten. (In certain areas use is made of this fact by driving the sheep into marshy infected parts before they are sold!) Soon the animal becomes weak and 'off colour' from fever, loses its appetite, and becomes anaemic. As the pallor increases, the skin becomes dry and scaly, and the wool, becoming brittle, falls out in patches. Later oedema develops, especially in the intermandibular space—hence the name 'bottle jaw'. Death may occur at this stage or earlier, and there may be blood-stained diarrhoea or constipation.

Cattle. The most characteristic symptoms are digestive disturbances. There may be marked constipation, with passage of hard and brittle faeces, or severe diarrhoea. The animal becomes dull and weak, and in chronic cases there is marked cachexia with gross anaemia. Severe prostration is especially common in calves.

Other Animals. Weakness, anaemia and emaciation are the most prominent symptoms.

CLINICAL FEATURES IN MAN

In general the disease runs a protracted course, with chronic ill-health and a long history of gastro-intestinal and hepato-biliary symptoms, which fail to respond to the usual methods of treatment. Frequently, however, there are no symptoms, or else very mild digestive disorders, and infestation is 'accidentally' diagnosed by the discovery of ova in the stools. In some cases, acute obstruction of the common bile-duct may be the first clinical manifestation of the disease (case 2). Fulminating cases with severe haemorrhage from the liver (case 1) have not been reported before. In most of the reported cases the disease presents two phases, viz. the stage of invasion, and later the stage of established infestation.

THE STAGE OF INVASION

In this stage symptoms are produced by the young flukes as they pass through the liver to the bile ducts. The symptoms, viz. abdominal pain and anaphylactic reactions, appear during the incubation period of 2-3 months, and disappear when the flukes reach maturity.

Abdominal pain, which may be extremely acute or only mild, is usually present and associated with a slightly enlarged, tender liver. It is accompanied by slight fever and toxaemic symptoms, e.g. anorexia, listlessness or simply being 'off colour'.

Anaphylactic reactions may occur during this stage. Pruritus and urticaria are common. Pulmonary symptoms are not infrequent, including a dry, non-productive spasmodic cough, asthma and pleural effusion. Pains in the joints and muscles occur occasionally. *Eosinophilia* is a constant and important finding, and often presents at a high level throughout the illness. Counts of 40% are frequent, and occasionally they reach 80%. Sometimes no eosinophilia occurs, or the count may gradually fall as the general condition deteriorates (this was noticed in our first case). On treatment with emetine the eosinophilia gradually diminshes, but may not reach a normal figure until 4 months later.

THE ESTABLISHED DISEASE

Various clinical pictures are found, the commonest being gastro-intestinal and hepato-biliary. These will be discussed under separate headings, but it must be remembered that a mixed clinical picture is not uncommon.

1. *Gastro-intestinal Manifestations*

Pain is almost invariably present at some stage of the disease. It is situated in the epigastrium and right hypochondrium. Often it is mild and described as indigestion, or it may simulate the pain of peptic ulceration. Occasionally it is very severe. (Biliary pain will be referred to below.)

Anorexia is common. *Nausea* and *vomiting* may occur, especially during attacks of severe pain. Although *constipation* may occur, *diarrhoea* is commoner. Occult blood is often present in the stools, and accounts for the anaemia, which is a common finding. Only one case of frank melaena has been reported, but it was not severe. Our first case was thus unique in that he had several massive gastro-intestinal haemorrhages requiring virtually exchange transfusions. Occasionally there is alternating constipation and diarrhoea.

2. *Hepato-biliary Symptoms*

A painful and tender diffuse enlargement of the liver is common in the early stages and may be associated with jaundice. Later the organ may become firm and irregular, with a nodular surface. The liver function tests in our first case gave grossly abnormal results.

Attacks of biliary colic, usually associated with jaundice, are common, and are mostly due to obstruction of the common bile-duct by the adult flukes themselves (case 2). These attacks are usually associated with distention of the gall-bladder, which may become palpable. Eight previous cases of operative removal of flukes from the common bile-duct have been reported, and in 4 of them stones were present in the gall-bladder, but not in the ducts.

Enlargement of the gall-bladder is usually due to obstruction of the common duct by flukes, but in our first case it probably resulted from the massive haemorrhage into the biliary system. Very rarely it may be due to acute cholecystitis with empyema.

Although *jaundice* may be absent, even in very ill patients, it is commonly present. It may last from a few

days only to a week and be accompanied by biliary colic, which is caused by mechanical obstruction of the common bile-duct or, if lasting over a prolonged period, by septic cholangitis. In the latter event it is often associated with rigors, high fever, and a leucocytosis, while eosinophilia is often present.

Splenomegaly associated with the hepatomegaly has been recorded on a few occasions (case 1). It may be due to infestation of the spleen or may be secondary to portal hypertension.

In the late stages the *abdomen becomes distended*, with dilated subcutaneous veins, ascites and other evidence of cirrhosis.

3. *General Symptoms*

Pyrexia is usually present in the stage of invasion, and later when there is septic cholangitis. A low grade fever with malaise, headache and anorexia may be the only manifestation of the disease. This may continue for a prolonged period. Later oedema appears and gradually the patient becomes increasingly cachectic and dies after the passage of several years.

Anaemia is a very common finding, and is due mainly to loss of blood into the bile-duct and bowel. *Leucocytosis*, with an *eosinophilia* of 20 to 40%, is a characteristic feature of the disease.

Various *other symptoms* which are less frequent, include cough and dyspnoea, headaches, insomnia and other nervous manifestations and allergic phenomena.

4. *Ectopic Sites*

Catchpole and Snow (1952) reported a case that presented with a granulomatous nodule in the abdominal wall overlying the right iliac fossa. This apparently 'migrated' to the right subcostal area within a week, and when it was excised it was found to contain an immature liver-fluke. They collected from the literature 24 other cases of ectopic siting, 14 in the subcutaneous tissues (9 in the right hypochondriac area), 2 in the peripheral veins, 2 in the appendix, and one each in the cerebrum, urine, portal vein, stomach, and peritonium. In one case the site was not mentioned.

5. *Halzoun*

In the Lebanon an infection known as Halzoun (because of suffocation) occurs after the ingestion of raw infected liver from sheep and goats. The parasites attach themselves to the mucosa of the mouth and pharynx, causing irritation, congestion and oedema, with respiratory embarrassment and sometimes associated anaphylactic symptoms.

DIAGNOSIS

Infestation by *F. hepatica* should be considered in all cases of obscure hepatic disease, particularly in areas where animal infestation is known to occur. Intermittent pyrexia, evidence of septic cholangitis and gastro-intestinal symptoms are features which should suggest the possibility of fascioliasis, particularly if these symptoms occur in epidemics.

DIFFERENTAL DIAGNOSIS

The main conditions which have to be considered in the differential diagnosis are:

1. *Gastro-intestinal diseases*, e.g. peptic ulceration, enterocolitis, and even carcinoma of the colon.
2. *Hepato-biliary diseases*, particularly cholelithiasis and also other causes of jaundice and hepatic enlargement.
3. *Other parasitic diseases:*

Amoebiasis, which is a common cause of diarrhoea and painful hepatomegaly in tropical and subtropical countries may present a very similar clinical picture. Since both amoebiasis and fascioliasis respond to emetine therapy, response to the drug cannot be taken as evidence in favour of either disease. It is possible that cases of fascioliasis have been mistaken for amoebiasis for this very reason.

Clonorchiasis produces a clinical picture which is indistinguishable from that of fascioliasis, but is almost entirely confined to the Far East, where up to 50% of the inhabitants in some districts are affected.

Fasciola gigantica, which is related to *F. hepatica* but larger (up to 75 mm. in length) and without a cephalic cone, is found chiefly in Africa and the Far East, where it may give rise to difficulties in diagnosis. Human infestation is very rare, but at least one case has been reported.

Schistosomiasis may affect mainly the liver and intestinal tract, and produce gross hepato-splenomegaly, but jaundice is uncommon. This disease also responds to emetine which, therefore, cannot be used as a therapeutic test.

Hydatid Disease of the liver is a fairly common cause of hepatomegaly in sheep-raising countries and, if the hydatids rupture into the larger intra-hepatic bile-ducts, the clinical picture may resemble that of *fascioliasis* very closely. Daughter cysts, like *F. hepatica*, may cause intermittent obstruction of the common bile-duct, with biliary colic, jaundice and a palpable gall-bladder, or rupture may be followed by secondary infection and a septic cholangitis.

Ascariasis may be responsible for mild toxaemic and digestive symptoms. Occasionally the parasite may enter the common bile-duct and cause biliary colic and jaundice associated with a palpable gall-bladder (we have encountered several such cases).

INVESTIGATIONS

1. *Eosinophilia* is an important finding in fascioliasis, and, in obscure cases, would suggest the possibility of a parasite infestation. It is usually more marked in fascioliasis than in other parasitic diseases.

2. *Ova in duodenal aspirate.* When the flukes are mature and passing eggs into the bile, the most reliable method of establishing the diagnosis is to find the ova in bile obtained by duodenal aspiration. In characteristic cases the bile contains brownish yellow floccules in which the ova are to be found. Possible sources of error are the unfertilized ova of *Ascaris lumbricoides* (which seldom inhabits the duodenum), and the ova of *Fasciolopsis buskii* (which is encountered in the Far East only.) This method of diagnosis was attempted in our first case but failed.

3. *Ova in the stools.* Ova may be recovered from the faeces in both human and animal cases. The faeces are

mixed with 50% glycerine and centrifuged. Other parasitic eggs float to the surface, but the heavy ova of *F. hepatica* sink to the bottom. The diagnosis in our first case was established by finding ova in the faeces and later in the connective tissue around the cystic duct. False positives may be obtained in subjects who have eaten infected livers that have not been adequately cooked.

4. *Intradermal and complement-fixation tests*

In the stage of invasion and while the flukes are maturing, which may take up to 3 months, the intradermal and complement-fixation tests may be of value. Although there appears to be some doubt about the reliability of these tests, Lavier and Stefanopoulo (1944) are quite emphatic about their value. Admittedly, their series was very small, but they had no false positives and no false negatives; 5 patients with fascioliasis were tested with intradermal and complement fixation tests, and all gave positive results for both tests. Four of the patients had had symptoms from 4 months to 10 years, and a 5th had had the disease for 10 years but had been cured by emetine treatment 6 months before the tests were made. The controls were 3 patients with Loa Loa infection, 1 with *Taenia saginata*, 1 with hydatid disease, 2 with serologically positive tests for syphilis, and 3 without fascioliasis who showed respectively an eosinophilia of 50%, a 'moderate' eosinophilia and a normal eosinophile count. All the controls gave negative intradermal and complement fixation tests to *Fasciola hepatica* antigen and all the patients with fascioliasis gave negative tests to antigens prepared from the other parasites. No antigen from Bilharzia was used in the tests; this may give false positive results, since *Fasciola hepatica* and schistosomes are closely related. The antigen was prepared by Farley's method as follows:

For the *intradermal test* fasciola obtained from the slaughterhouse were dried and powdered and 1 g. of the powder was kept in 100 c.c. of physiological saline for one day at 37° C and the supernatant fluid filtered through a Seitz filter. For use it was diluted 1 : 4 or 1 : 5 or more. The antigen gave an immediate intradermal reaction; after 3-5 minutes a wheal with pseudopodia appeared, which reached 3 cm. in diameter, erythema appearing later. In 1-2 hours both erythema and wheal had disappeared, but infiltration of the skin and tissues round the reacting area persisted for 4-6 hours. After 24 hours the reaction had disappeared completely. Delayed reactions were not observed but some patients who gave strongly positive reactions showed delayed phenomena such as generalized urticaria or pain and swelling of neighbouring joints and 2 showed nausea, profuse diarrhoea lasting several hours, and shock. Subjects sensitive to foreign proteins may show false positive reactions, but these reactions are usually surrounded by a small erythematous halo not more than 1 cm. in width, which disappears very quickly.

For the *complement-fixation test* 1 g. of dried and powdered *Fasciola hepatica* was dissolved in 100 c.c. of absolute alcohol, and kept at 37° C for 24 hours, filtered and evaporated until, when it was evaporated to ⅓rd of its volume, it (usually) became cloudy. Absolute alcohol was added to make up the initial volume and the precipitate disappeared. This final solution formed the antigen. It was usually effective at a dilution of 1/20.

TREATMENT

Prophylaxis

1. Eradication of the intermediate host. The snails may be destroyed by burning the vleis and marshes in the dry season. This is simple and cheap but not entirely successful. A better but more laborious and expensive method is to add some chemical such as copper sulphate to the water. The details of this method can be found in any standard veterinary text-book.

2. Infection can be avoided by not eating watercress etc., and by boiling or filtering all drinking water that might be contaminated.

Curative Treatment

Animals. Pure carbon tetrachloride was formerly used for sheep, but was too toxic for cattle; now hexachlorethane is used for both sheep and cattle. It is recommended that sheep recently removed from pastures should have a second course of treatment after 4-6 weeks, on the supposition that the young flukes in migration may not be destroyed.

Man. The fluke normally lives for 9 months (though it may live up to 5 years), and mild infestations with minimal symptoms may be self-limiting, provided that re-infestation does not occur.

Emetine is specific and should be given as soon as the diagnosis is made. Gradwohl and Kouri advise consecutive daily subcutaneous injections of 4 cg. till a total equivalent to 0·5 cg. per kg. of body-weight has been reached. A second course of treatment has not been recommended in man, but our first patient failed to respond to a single course of emetine, and yet made a dramatic recovery after the second course, which was given 4 months later.

Surgical treatment is not necessary, provided the diagnosis can be made without operation. If, however, diagnosis is only made at operation during exploration of the common bile-duct, the flukes should be removed and the duct drained by a T tube. This should be left *in situ* for varying periods depending on the degree of inflammation and fibrosis of the duct. The gall-bladder in these cases should be opened, for it may contain flukes occasionally, but it need not be removed unless it contains stones or is inflamed.

Operative intervention for haemorrhage is of no avail, as illustrated by our first case.

CASE REPORTS

Case 1

J.C L., a European boy aged 3 years and 4 months, was admitted to the Groote Schuur Hospital, Cape Town, on 7 March 1953. He had been resident in the Belgian Congo for 15 months, and became ill 9 months before admission. During these 9 months the following were the main clinical features:

1. Recurrent febrile episodes (100-102° F), with upper abdominal pain, vomiting and diarrhoea.
2. Progressive anaemia and listlessness and increasing pallor but weight stationary at 30 lb.
3. White blood cells varying (in 5 counts) between 12,600 and 18,900 per c.mm., with eosinophiles varying between 17% and 32%.

4. Hepatomegaly, first detected 4 months before admission.
5. Two episodes of malaena—on each occasion a blood transfusion of 300 ml. had to be given.

The salient features on admission were as follows:

1. Gross pallor (haemoglobin 5·5 g.%).
2. Pyrexia of 100-104° F.
3. Gross hepatomegaly, with 3 nodules on the surface of the right lobe of the liver.
4. Gall-bladder palpable.
5. Tip of spleen palpable.
6. Signs in the lungs suggestive of pneumonia.
7. White-cell count 16,700 per c.mm., with 24% eosinophiles.
8. Blood in the stools.
9. Grossly abnormal liver function tests (thymol turbidity 5, thymol flocculation 4, colloidal gold 4).

Stools were sent for microscopical examination and culture, and on two occasions ova of *F. hepatica* were isolated.

Progress in hospital was briefly as follows:

1. A blood transfusion (500 ml.) was given immediately and the boy was put on emetine, gr. 1/5 daily for 10 days, and antibiotics. No dramatic improvement followed, but the malaena ceased, and the gall-bladder became impalpable. Ova of *F. hepatica* disappeared from the stools after 1 week and were never found again.

2. For 6 weeks he had recurrent episodes of high fever, accompanied by severe malaena, jaundice and enlargement of the gall-bladder. These lasted for 3-4 days, during which time he had to be transfused (300 to 1,500 ml. per transfusion). The episodes were succeeded by remissions of 1-2 weeks.

3. Six weeks after admission *laparotomy* was performed (J.H.L.) to look for flukes in the common bile-duct and a possible source of bleeding (e.g. Meckel's diverticulum) in the small bowel. The liver was found to be grossly enlarged, with marked peri-hepatitis and studded with numerous small necrotic 'abscesses' (material from these contained necrotic tissue and foreign-body giant-cells, but no flukes or ova). The common bile-duct was thickened and slightly distended but on exploration no flukes or calculi were found. The gall-bladder was distended and, although not obviously diseased, was removed. (On histological examination by Dr. Golda Selzer this revealed an extensive intense inflammatory reaction in the connective tissue surrounding the cystic duct. There was a diffuse infiltration of plasma cells, macrophages and eosinophils and, in addition, localized foci of pseudo-tubercles around large ova of *F. hepatica*—Fig. 4. The gall-bladder showed a thickened serosa and a limited inflammatory-cell infiltration of its wall.)

The patient seemed to improve after the operation, but within a week the episodes of fever with jaundice and melaena recurred and now blood also poured from the T-tube that had been inserted in the common bile-duct. Further transfusions were required on several occasions.

A cholangiogram done about 6 weeks after the operation, during an unusually prolonged remission, revealed no abnormality of the biliary tree, and the T-tube was removed. Soon afterwards there was a severe relapse, which was followed by several more during the next few weeks.

4. Nine weeks after the cholecystectomy it was decided to perform another laparotomy in a desperate attempt to get to the source of bleeding, which had become practically uncontrollable. At this operation (J.H.L.) several large necrotic areas in the liver were explored, but the exact source of the bleeding could not be found. The liver was riddled with 'abscesses', some of which projected from its surface.

The child was now *in extremis*. Cortisone was given with no effect and he required many more blood transfusions. By this time he had had 15 transfusions amounting to approximately 6 litres; in one period of 3 weeks no less than 3 litres were given. His blood volume was only 1,500 ml. and on one occasion a single transfusion of 1,500 ml. was required.

5. When he had been in hospital for 4 months, and the condition appeared to be hopeless, he was given another course of emetine, which was followed by immediate and dramatic improvement, cessation of all bleeding and rapid recovery.

6. He was discharged in a fit condition 1 month later, the only abnormality being an enlarged, firm, irregular liver. When last seen in July 1956 (3 years later) he was quite well.

Comment. This case is unique because of his young age, the severe haemorrhage, and the fact that a second course of emetine was required to effect a cure.

Case 2

E.Q.G., a European male aged 45, was referred by Dr. G. J. Budow, of Goodwood, with a history of an attack of severe biliary colic in January 1955, followed by jaundice with pale stools and dark urine for 14 days. In March he again had severe biliary colic followed by jaundice lasting 3 days. There had been no febrile illness or urticaria before the first attack. He was a commercial traveller, but had not been outside the Cape Peninsula for several years and had never drunk water from any stream or pond, but admitted to being very fond of lettuce and watercress.

A clinical diagnosis of stone in the common bile-duct was made and operation advised. Cholecystogram and barium meal were normal.

At operation (W.W.) the liver was normal, but the gall-bladder was distended to about twice the normal size. It contained no calculi and was not thickened or inflamed. The common bile-duct was white, thickened and obviously inflamed when it was explored. Four flat leaf-like structures were removed, which Dr. Selzer identified as liver flukes (*F. hepatica*). There were no stones and a probe passed easily into the duodenum. The gall-bladder was opened, but it contained no flukes or calculi. The gall-bladder and common bile-duct were drained, the latter by a T tube, which was removed after 3 months.

Emetine hydrochloride, gr. 1, was given daily by subcutaneous injection for 10 days. On the 8th day a solitary ovum (Fig. 2) was recovered from the bile, and so a second course of emetine was given after an interval of 10 days. No further ova were recovered.

The patient made an uneventful recovery and has had no further symptoms.

Comment. This is the 9th reported case of operative removal of *F. hepatica* from the common bile duct.

SUMMARY

The distribution, clinical features, diagnosis and treatment of infestation of animals and man by *Fasciola hepatica* are described, and 2 cases of human infestation recently encountered are reported.

It is pointed out that animal infestation is common in certain summer-rainfall areas of South Africa, and it is suggested that human infestation may be less rare than is generally believed.

In endemic areas the condition should be suspected in all persons who present with obscure hepato-biliary disease, especially if associated with anaemia, eosinophilia or bleeding from the intestinal tract.

The diagnosis can be established by recovery of ova from the duodenal aspirate or stools of the patient. Complement-fixation and intradermal tests are available, and may be of some value.

Treatment with emetine is specific, and usually highly successful, but 2 courses at an interval of 1-2 months may be necessary in recently infected cases. Surgical treatment should rarely be necessary, but may be required for obstruction of the common bile-duct.

We wish to thank the following:

Dr. Helen Brown and Dr. G. J. Budow, who referred cases 1 and 2 respectively.

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Drs. G. Selzer and A. Kipps, for identifying the ova and flukes, and for the pathological reports.

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A REVIEW OF 463 EARLY ECTOPIC PREGNANCIES OPERATED UPON AT THE GROOTE SCHUUR HOSPITAL*

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As everybody who has come to grips with statistical figures knows, they can be the most unrelenting of tyrants or the most amusing of servants. The results of an encounter with the figures involving ectopic pregnancies are presented. During the 5 years 1951-55 there were 12,682 admissions to the gynaecological wards of the Groote Schuur Hospital, Cape Town, of which 463 were early ectopic pregnancies, representing an incidence of 3·6%. The pregnancies were tubal in situation in all but one case, in which the implantation was in the right ovary.

Age and Race Incidence

Most patients in the series of 463 cases of early ectopic gestation were between the ages of 25 and 40 years. There were only a few cases under the age of 20 years. The youngest was 17 years and the oldest 44 years. The majority of the 463 patients belonged to the Coloured race—58%. The numbers were the same (21%) for both the Bantu and White. (The European and Coloured populations of Cape Town are approximately equal, and the Bantu population relatively small.)

AETIOLOGY

Although a great variety of causes—for example, chronic pelvic infection, congenital abnormalities of the tube, extratubal swellings, tubal spasm, transmigration of the ovum and increased receptiveness of the tubal mucosa—have been suggested, tubal inflammatory disease is probably the greatest single aetiological factor. A history of pelvic inflammation was elicited in 126 cases (27%) but histological confirmation was obtained in only 61 cases (13%). In 3 of the latter the salpingitis was found to be tuberculous in origin. As salpingitis is frequently a bilateral disease, it was not surprising to find that repeated tubal pregnancy occurred in 29 cases (6·3%). The use of sulpha drugs and antibiotics has greatly reduced the incidence of pelvic inflammation. In 1946 approximately 62% of all cases seen at the gynaecological out-patient department were diagnosed as pelvic infection; this figure had dropped to 24% in 1955. A sharp fall in the frequency of ectopic pregnancies might therefore be anticipated. This conclusion differs from that of Johnson and Post (1954), who state that the use of antibiotics has been responsible, in fact, for an increased incidence of tubal pregnancies. These authors consider that the antibiotics arrest the inflammatory process before

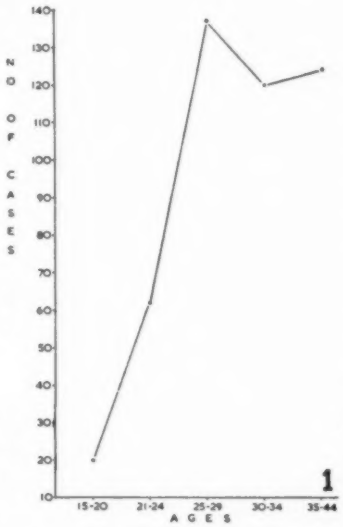

Fig. 1. Ectopic pregnancy: age incidence.

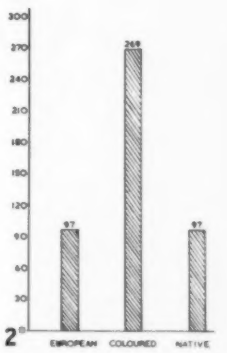

Fig. 2. Ectopic pregnancy: race incidence.

* A paper read at the South African Obstetrical and Gynaecological Congress, Durban, July 1956.

complete occlusion results, thus predisposing to tubal implantation.

The graph in Fig. 3 shows for each of the 10 years 1946-55 the number of cases of ectopic pregnancy admitted to the gynaecological wards of the hospital, as compared with the total admissions to those wards and the population of the Cape Town municipality, and the fall in the 10 years in the percentage of cases of pelvic infection in the gynaecological out-patients. The graph shows that there has been a steady rise in the number of ectopic pregnancies, interrupted in 1953 and 1954. In 1955 an additional ward was made available to the gynaecological department and this appears

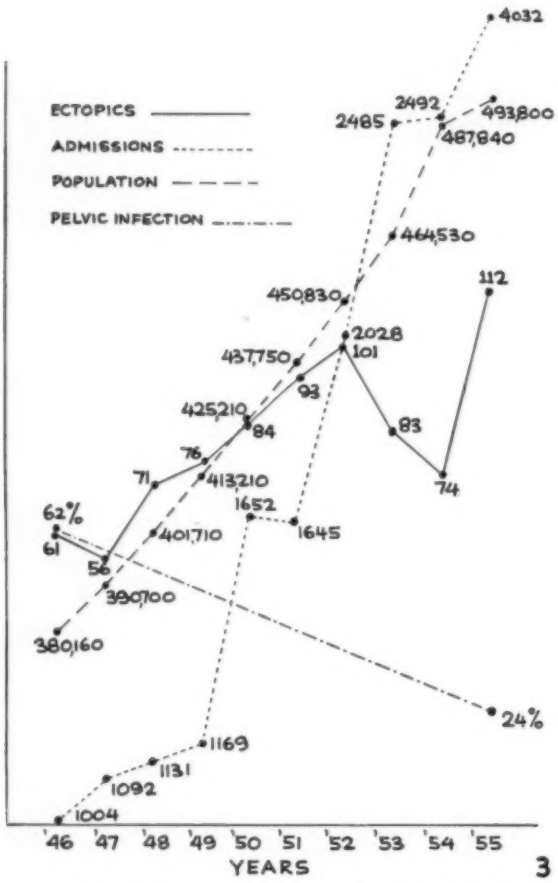

Fig. 3. Ectopic pregnancy: annual number of cases admitted, etc.

to account for the increase in 1955. In 1946, 1,004 cases (including 61 ectopics) were admitted to the gynaecological wards, as compared with 4,032 (including 112 ectopics) in 1955. The population of Cape Town has also increased by 113,640 (30%) during the last 10 years. It therefore seems that the increased number of tubal pregnancies in Cape Town has kept in trend with the rise in both the population and the number of admissions.

SYMPTOMS

The main symptoms can be summarized as follows:

Abdominal pain	95%
Shoulder pain, including subcostal	28%
Amenorrhoea	82%
Vaginal bleeding	74%
Gastro-Intestinal symptoms	42%
Bladder symptoms	18%

There is general agreement that abdominal pain, amenorrhoea, and vaginal bleeding are the important triad. Practically all the patients complained of *abdominal pain* but it varied greatly in severity and site. In 28% of cases pain was referred to the shoulder tips and this was found to be a valuable diagnostic guide. Localization of the pain did not necessarily indicate the side of the ectopic pregnancy. A history of *amenorrhoea* was present in 82% of cases. It was interesting to note that 10 cases of tubal pregnancy occurred during the amenorrhoea of lactation. *Vaginal bleeding* occurred in 74% of cases. In many instances the bleeding resembled the onset of normal menstruation but continued as persistent dark spotting. Profuse bleeding was seen in 29 cases. *Gastro-intestinal symptoms* were surprisingly common; 42% of cases complained of nausea, vomiting and diarrhoea. The importance of *bladder symptoms* was first recognized by MacVine and Lees (1949). They usually take the form of dysuria and frequency. In this review 3 cases presented with urinary retention.

DIAGNOSIS

An accurate pre-operative diagnosis was made in 65% of the 463 cases of ectopic gestation. The wrong diagnoses were as follows:

Tubo-ovarian masses	17·4%
Fibroids	4·2%
Abortions	4·0%
Ovarian cysts	3·7%
Appendicitis	2·1%
Others	3·6%

In 22 cases laparotomy was performed following an erroneous diagnosis of ectopic pregnancy.

Routine haemoglobin estimations were made; in 72% of cases the reading was under 10 g.%. A falling haemoglobin reading was found to be a valuable diagnostic guide. Frog tests were found to be of minimal help in diagnosis. Colpopuncture should not be used as a routine measure. The prolonged anaesthetic is an added risk to a shocked patient and occasionally false positive and false negative results are encountered. In this review colpopuncture was used as an aid to diagnosis in 72 cases. It should be reserved for cases where the diagnosis is in doubt.

TREATMENT

In this series there were 2 deaths—a mortality rate of 0·4%, which compares more than favourably with

those found in the literature. One of the fatal cases was admitted in a moribund condition, and died before treatment could be administered. The other was a 28-year-old patient who was treated by right salpingectomy. After an entirely satisfactory post-operative course she suddenly collapsed and died on the 7th post-operative day; post-mortem examination revealed a massive pulmonary embolus.

There seems little doubt that spontaneous cure occurs in some cases and a few gynaecologists still practice conservative treatment of ectopic gestations. At the Groote Schuur Hospital operation is undertaken in every case as soon as the diagnosis of tubal gestation is made. Delay in pre-operative resuscitative measures is seldom justified, because these patients are actively bleeding and the surgeon's first duty is to arrest the haemorrhage. The patients improve dramatically once the haemorrhage has been controlled. Blood transfusions are given when indicated, which was in 82% of the cases in this series.

The following were the operations performed:

Right salpingectomy	187 cases
Left salpingectomy	176 cases
Salpingo-oöphorectomy	87 cases
Wedge resection of uterus	11 cases
Oöphorectomy	1 case

Thus salpingectomy was performed in 97% of the cases. The incidence of salpingectomy on the right and left sides was roughly the same. No attempts were made to conserve the fallopian tube. Total salpingectomy is advocated because the risk of recurrence in the same tube is high—MacVine and Lees (1949). The operation of tubal conservation should be reserved for cases where the opposite tube has already been removed or is diseased.

All too frequently the ovary on the affected side is removed with the fallopian tube. This is an unjustifiable practice, despite the views expressed by Jeffcoate,[3] who suggests that salpingo-oöphorectomy will improve the conception rate in patients who have suffered from atubal pregnancy. Jeffcoate makes no attempt to prove this theory and no known figures to date substantiate his statement. It is generally possible to dissect the ovary free from the surrounding adhesions and thereby save it.

Some authorities recommend hysterectomy as the treatment of ruptured interstitial pregnancy but wedge-resection of the uterus was found to be a satisfactory and adequate form of treatment in the 11 cases in this review.

In one patient a bilateral ectopic pregnancy was discovered. It is therefore essential for the surgeon always to inspect the opposite adnexae.

Post-operative complications were surprisingly few. In this series there were 16 cases of post-operative complications involving the lungs. In the light of recent knowledge, the majority of these were probably cases of pulmonary embolus.

SUMMARY

463 cases of early ectopic pregnancy were admitted to the Groote Schuur Hospital in the 5 years 1951-55. The mortality rate was 0·4%. In order to achieve a low mortality, early diagnosis, early admission to hospital, together with rapid replacement of blood lost and adequate surgery are essentials.

I wish to express my appreciation to Prof. James T. Louw, Professor of Obstetrics and Gynaecology, University of Cape Town, for his encouragement and helpful criticism. I also wish to thank Dr. N. G. Cloete, Superintendent of Groote Schuur Hospital, for permission to publish this work.

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SOUTH AFRICAN ORTHOPAEDIC CONGRESS

The 5th Congress of the South African Orthopaedic Association* was held in Durban on 13-18 August 1956. The meeting was presided over by Mr. C. T. Moller, the President of the Association. Distinguished guests from outside South Africa included Sir Walter Mercer, President of the Royal College of Surgeons of Edinburgh, Dr. Steele F. Stewart from Honolulu, and Mr. F. Dwyer from Liverpool.

The meeting was a success from both an academic and social point of view. It was attended by 30 orthopaedic surgeons practising in South Africa and Rhodesia. A visit was made to the Umlazi Orthopaedic Tuberculosis Hospital and members were entertained at several social functions. The annual orthopaedic dinner was marked by a tribute to Mr. F. P. Fouche, the doyen of orthopaedic surgery in South Africa, who was presented with a silver salver bearing the facsimile signatures of all orthopaedic surgeons in the Association, in honour of his 70th birthday.

SYMPOSIUM ON ORTHOPAEDIC TUBERCULOSIS

Sir Walter Mercer opened the symposium by discussing the *diagnosis and treatment of tuberculosis of the hip joint*. He showed slides depicting the radiological appearances of the intra-articular and extra-articular types of hip tuberculosis, and emphasized the difference in diagnosis and treatment of these types. In the synovial types, a biopsy assists in the diagnosis and helps in treatment by decompressing the tension in the joint. Rest and antibiotics also assist in the treatment. The action and dosage of the various chemotherapeutic substances such as PAS, INH, and streptomycin were mentioned. In the extra-articular types of tuberculosis of the hip, rest by leg traction and eradication of the bony focus was advisable, and the treatment of sinuses by chemotherapy and arthrodesis of the hip by Britain's method were mentioned. A movie film showing the speaker performing a Britain's arthrodesis by the posterior approach was presented.

Mr. N. M. Thompson (Pietermaritzburg) described his results with *excision of the head and neck of the femur* in 15 cases of tuberculosis of the hip in adults who had not responded satisfactorily to rest and chemotherapy. Most of these cases were acute, with pyrexia, pain, inability to walk, and sometimes discharging sinuses. The hip is opened by the anterior or lateral approach; sinus tracts and all affected soft tissues are excised, the neck of the femur is osteotomized, and the head is scooped out. No attempt is made to dislocate the joint. The operation gets rid

* Report (1956): S. Afr. Med. J., **30**, 827.

of a great deal of tuberculous infected tissue; it opens up the tissues to chemotherapeutic agents, and puts the affected acetabulum at rest, because weight bearing is now lateral to the acetabulum. A loose-fitting Thomas caliper is prescribed and gluteal exercises are practised. The patients, however, soon discard the caliper and walk with one stick. They can walk up to 6 miles if capable of re-education, but heavy patients do not walk so far. Passive movements are usually full after the operation, but there is no active abduction. Shortening is usually 1½ inches. The speaker said he believed this operation should be done in all elderly adults in whom conservative treatment had failed.

Mr. A. C. Boonzaier (Johannesburg) presented his results of *central dislocation-arthrodesis* as a radical treatment of tuberculosis of the hip joint. He had operated on 25 cases of severe tuberculosis of the hip, of which 18 were adults. There was no operative mortality and no abscesses or sinuses; and all the wounds healed primarily. Fourteen cases were healed solidly by fusion clinically and radiologically at the end of 12 months. Six cases were immobile clinically but showed evidence of non-fusion radiologically, although they were symptom-free; 2 cases had slight movement of the hip but were painless and ambulant. The other 3 cases had not returned for a final assessment.

The operation is performed as soon as the patient's general condition is satisfactory and has been improved by blood transfusion and antibiotics. Flexion contracture if present is first corrected by leg traction. A wide Smith-Petersen or antero-lateral exposure is employed, sectioning the tensor fasciae latae a handsbreadth below the trochanter, osteotomizing the lesser trochanter and detaching the muscles from the greater trochanter. Every single bone-abscess in the femur and acetabulum is ablated, and the entire capsule and all granulation-tissue is excised. The sclerosed bone of the head, neck and acetabulum is broken down, but the calcar femorale is left intact. The head and neck are shaped by osteotomes, and Charnley's reamers are used to drill a close-fitting hole in the acetabulum. If the acetabulum is ruined by the disease or the debridement, a hole is made in the pelvis a little more proximally. The head and neck are centrally dislocated into the pelvic hole, and multiple boiled cadaveric bone-chips are laid across the fusion site. A full spica is applied for 3 months and another spica from the nipple line to above the knee for another 3 months. Weight bearing can be allowed in this latter spica if radiological union appears to be progressing satisfactorily. Otherwise non-weight-bearing should be practised for a total of 6 months. The triad of PAS, streptomycin and INH are given for 12 months.

It is felt that this debridement and fusion operation should be practised in all adults and children with grossly destroyed tuberculous hips.

X-ray slides were shown of nearly all the cases operated upon.

Mr. C. J. Kaplan (Durban) presented his observations on *local therapy in skeletal tuberculosis*. He illustrated by means of slides and X-rays the additional efficiency of parenteral and intra-articular antibiotics and chemotherapy, in contrast with the previous treatment of rest, diet and sunshine alone. The former periods of hospitalization extending over 3 or 4 years was more than halved, and sinuses and secondary infection were almost completely eliminated.

Streptomycin, ½-1 g., was given intramuscularly every day for 1 month, and then every 3rd day for the duration of stay in hospital.

PAS is no longer used as a routine therapy because it tends to heal tuberculous lesions by fibrosis instead of resolution without scarring.

Isoniazid (INH) is given in doses of 8-16 mg. per kg. of body weight and is supplemented with vitamin B to prevent peripheral neuritis.

Other cases were treated in addition by intra-articular injections of Streptohydrazid (a combination of streptomycin and INH) twice weekly for 3 months. X-rays were shown of tuberculous hips and knees in which painless function and useful mobility were regained after this method of treatment.

Mention was made of treatment of Pott's spine, with abscesses, by means of drainage and continuous drip-perfusion of the cavity with Streptohydrazid in saline. A case of proved tuberculosis of the flexor tendon sheaths of the fingers had also been successfully treated by this drip-perfusion method.

Mr. J. J. Commerell (Cape Town) described the *domiciliary treatment in orthopaedic tuberculosis* as practised in Cape Town and its environs. During the last 6 years 410 cases had been treated on Thomas frames and other types of immobilization in their homes. Of these 376 were cases of tuberculous involvement of spine, hips and knees.

Most of these cases were under 28 years of age. Older cases were difficult to treat at home because there was usually no one to attend to them.

District nurses visited the patients regularly to give them their streptomycin injections and INH tablets, and to inspect their frames. In 218 of these cases treatment had been continued until the disease had become arrested. The average length of treatment was 16 months for spinal tuberculosis, and 14 months for hip tuberculosis.

Over the 6-year period 4 patients died of various causes and 126 required admission to hospital for various complications or inability to continue treatment at home.

There now only remained 28 cases on Thomas frames at their homes, which proved that a large back-log of skeletal tuberculosis had been cleared up by means of early diagnosis and adequate chemotherapy and rest.

Dr. H. G. Houghton (Durban) spoke on the recent trends in tuberculosis therapy, with special emphasis on pulmonary and cerebrospinal pathology. He made the following notable points:

The chronicity at the end of treatment resulted in 10% of patients with positive sputa.

PAS is no longer given as a routine.

INH is given in dosage of 15 mg. per kg. of body-weight, daily. Streptomycin 1 g. per day is also given. 10% of cases become resistant to INH and new drugs such as Neotocide and Dipasic are being tried.

Today 2,000 cases of pumonary tuberculosis in Durban are being treated outside hospital as ambulant patients.

The value of bed rest is being challenged.

The length of treatment is usually 2 years.

Calcified foci in the lung should be treated.

THE DURBAN MYSTERY OUTBREAK RESEMBLING ICELANDIC DISEASE

Mr. R. C. J. Hill (Durban) opened the discussion by describing the epidemic of a disease resembling poliomyelitis which occurred in Durban in March 1955. The main features were a pyrexial illness followed by weakness of the trunk and limbs, clonic contractions of muscles, sensory changes, and relapses of these symptoms. The condition persisted with periodic relapses for many months and was often associated with psychological changes in the patient.

The cases included 98 members of the nursing staff of the Addington Hospital, Durban, and 45 civilian patients. In this outbreak 12 nurses and 4 civilians had become permanently incapacitated. Only 12 of the patients were males and the vast majority were between 18 and 25 years of age.

Dr. R. W. S. Cheetham (Durban) divided the disease into a prodromal, an acute and a convalescent phase. He demonstrated a nursing sister who was still incapacitated. She had patchy loss of sensation in the lower limbs and developed clonic contractions on moving the left arm and leg against resistance. Weakness of the abdominal and back muscles was almost a constant feature of this malady.

Dr. G. A. Joubert (Pathologist, Springfield Hospital, Durban) described the routine and specific methods of investigation adopted during the epidemic. In his opinion circumstantial evidence pointed rather to a virus than to a toxicological agent.

Dr. G. D. English (Medical Officer of Health, Durban) felt that the disease had some connection with the endocrine organs and hoped that the examination of urinary ketosteroids might be useful.

Dr. R. Percy-Lancaster (East London) had seen 4 cases in East London, and described the progress of one case in detail. He noted that in most cases the left side of the body was involved. He described the psychological aspects and the treatment that is sometimes necessary for these symptoms.

OTHER PAPERS

Osteochondritis Dissecans

Mr. R. Percy-Lancaster (East London) discussed this condition as it affects the ankle and the elbow joints. He showed X-rays of

these affected joints before and after operative removal of the loose fragments. The question of treatment led to some discussion. *Mr. J. M. Edelstein* (Johannesburg) mentioned the rarity of reported cases affecting the capitellum, and *Sir W. Mercer* indicated that unseparated areas in children would re-unite if immobilized for 6 months. If the fragment was separated it should be removed. *Mr. B. Polonsky* (Johannesburg) mentioned a case in which he had removed the affected head of radius.

Recurrent Sprains of the Ankle Joint

Mr. J. F. P. Mullins (Durban) dealt with the partial diastasis that occurs at the inferior tibio-fibular joint after a severe sprain of the ankle. Patients with this condition usually have a history of injury leading to recurrent sprains of the ankle. There is a sense of instability and chronic pain after weight bearing, and the ankle tires easily. X-rays usually show slight widening between the medial malleolus and talus, and sometimes a positive inversion or eversion test. The most valuable sign is a click in the ankle which is elicited by rocking the talus from side to side with the ankle held in the neutral position. The degree of rocking indicates the degree of instability.

He had operated on a large number and had full details of 25 of these cases. Some had been followed-up for 6 years, and all were relieved of their disability. The operation consists of screwing the tibia and fibula together, using a Johanssen lag-screw. The patient is kept in bed for 2 days and then allowed to walk. No plaster or compression bandage was necessary. The oldest case treated was 65 years of age, who had had symptoms for 25 years. He found wiring of the bones or bony fushion of the inferior tibio-fibular joint unsatisfactory.

Mr. T. B. McMurray (Cape Town) said that a good test of instability of this joint was to force dorsiflexion of the ankle, which invariably gave rise to pain because it caused the inferior tibio-fibular joint to spread apart.

Prof. C. Lewer Allen (Cape Town) indicated that he preferred fusion of the tibia to the fibula 2 inches above the ankle joint by cross-flapping osteo-periosteal grafts across the gap after removing the fibrous material between the bones.

Mr. C. Kaplan (Durban) felt that despite the normal movement which occurs at this joint, results seem to indicate that this operation is sometimes necessary, but he would prefer to immobilize the ankle in plaster.

Treatment of Flat Feet in Children

Mr. A. J. Helfet (Cape Town) had treated 500 cases of flat feet in children and foot-strain in adults by means of heel-seats. He depicted by means of diagrams and photographs how the usual arch-supports and wedged heels are ineffective in treating flat feet. The patient's heel merely slides laterally and deforms the outer stiffening of the shoe and wears down the heel. The usual complacent expectation that nature will correct most flat feet should be discarded; a vistit to any foot clinic or recruiting depot will show this. He had devised a heel-seat made of acrylic, which fits snugly around the heel and the sides of the os calcis, and corrects any valgus tendancy. It can be worn in any type of shoe, football-boot, gum-boot, sandal, tennis-shoe, or slipper. It holds the heel in a vertical position and prevents the usual deformity of the shoe. It therefore saves expense on shoes, is comfortable, and need not be replaced more frequently than once or twice a year. It should be worn for about 2½ years.

Mr. J. J. Commerell (Cape Town) suggested that everted feet were due to a tight tendo achillis and that the relief obtained by heel-seats in these cases seemed to be due to the raising of the heel and therefore release of tension on this tendon.

Mr. C. T. Moller (Johannesburg) asked if the tight tendo achillis was cause or effect in these cases. The Whitman's valgus-brace served a similar function to that of the heel-seat, but most technicians could not seem to fit the patient accurately.

Mr. R. C. J. Hill (Durban) noticed that when adolescent girls start wearing high heels they walk better and feel better, because of relief of the tight tendo achillis.

In reply to *Mr. F. P. Fouche* (Johannesburg) Mr. Helfet said that it did not matter what the cause of the flat feet was. Any child who persists in walking on the inner borders of his feet can be treated by this method. The shape of a foot can be moulded by this method, just as the Chinese women used to mould their feet by binding them tightly.

The Management of Scoliosis

Mr. F. J. Hedden (Johannesburg) stated that, although the management of scoliosis was still controversial, it had in recent years gradually progressed along certain sane and definite lines.

He outlined the clinical examination of a case of scoliosis of the spine and demonstrated the usefulness of X-rays, particularly of 'bending' films.

Idiopathic scoliosis was the commonest (80%). Other causes of scoliosis were poliomyelitis (5%), neurofibromatosis (2%), congenital (less than 2%), thoracogenic (less than 2%), Morquio's disease (1%), Friedreich's ataxia, spastics.

He classified scoliosis as either functional or structural.

He then described the various types of idiopathic scoliosis: Lumbar scoliosis occurs in 26% of cases and the prognosis is good. Thoraco-lumbar scoliosis occurs in 8% of cases and also has a good prognosis. Thoracic scoliosis occurs in 43% and is the most important group, giving rise to the largest curves and producing the worst deformities. It occurs at 3 periods of rapid growth and can be described as either infantile, juvenile, or adolescent. The combined thoracic and lumbar scoliosis occurs in 23% of cases and the prognosis is good. He discussed treatment in all the different types of scoliosis, and mentioned the roles played by physiotherapy, corrective plaster beds, plaster jackets and spinal supports, and correction followed by spinal fusion. He described the method of correction and fusion in detail. He employs the Risser turn-buckle jacket to obtain correction of the scoliosis. He then tries to obtain a sound bony fusion of the whole of the primary curve, if possible in a one-stage operation, by means of refrigerated bone-bank bone. The patient is kept recumbent for 6 months in the Risser jacket after fusion, and is then allowed up in a polythene jacket or if necessary a Milwaukee brace. One year after operation, films of the primary curves are taken to exclude pseudo-arthrosis and, if the results are satisfactory, all support is abandoned.

His conclusions were that 5% of cases of idiopathic scoliosis need early fusion and correction, mainly in the thoracic type at the age of 10 years, because of the bad prognosis. In 50% of paralytic curves operation is needed because of instability, symptoms or deformity. Early fusion is especially indicated in the thoracic type.

Nearly all cases of neurofibromatosis need early operation, but operation is only rarely necessary in the congenital type. He felt that scoliosis should be dealt with in a specialized clinic where adequate facilities and time are available.

Malignant Bone Tumours

Dr. M. Findlay (Durban) discussed the diagnosis and treatment of bone tumours. X-rays and slides were shown of the various tumours and the following points were mentioned:

In osteoclastoma 50% had a favourable result if properly treated, 35% tended to recur and 15% became malignant. The treatment of osteoclastoma is still controversial. Some are of the opinion that irradiation after curettement causes malignancy, but Dr. Findlay disagrees and considers it the best line of treatment.

In osteogenic sarcoma it is best to irradiate first (with supervoltage if possible) and amputate afterwards. The 5-year cure rate ranged from 10% to 23%.

Arteriography was mentioned as a means of differentiating an inflammatory from a neoplastic bone lesion, or in diagnosing malignancy at an early stage in a benign tumour. Arteriograms were shown depicting the various differentiating features, such as 'pallor', branching of vessels and arterio-venous communications.

Dysplasias of Tendons of Feet as a Cause of Congenital Deformities

Dr. Steele F. Stewart (Honolulu) compared the development of the foetal foot in the macaque, the gorilla and the human. He is of the opinion that the human foot is more primitive than the monkey's foot, and that it stands at the base of the primate tree.

If the tendons differentiate and develop earlier than normal in a human foetus, arthrogrypotic and other deformities are more likely to be present at full-term. The longer it takes for the foetal development of the tendons in the foot, the lesser are the gradations of these foot deformities at birth.

In club-foot deformities there is a definite expansion of the tendo achillis, which attaches more medially on the os calcis than in the normal foot. The tendo achillis consists of a flat superficial band formed from the inner head of gastrocnemius, and a flat volar or deeper band formed from the outer head of gastrocnemius. These closely-applied flat bands can always be seen and separated. It is the deeper band from the outer head of gastrocnemius which attaches to the medial surface of the os calcis in the club foot. This observation has led him to devise a new operation to correct club-foot inversion of the heel. He detaches the deeper band from its insertion into the os calcis, and finds that the heel can immediately be everted more easily. He then tenotomizes the superficial band of the tendo achillis to gain length, and inserts the distal end of the detached deep head into the distal end of the cut superficial head. In this manner a more direct and straight pull on the os calcis is obtained.

Other tendon anomalies in club foot are corrected at the same operation. The erroneous dorsal expansion of the peroneus brevis is severed and transplanted volar-wards. Also the anomalous bands of peroneus longus in its plantar tunnel are severed to allow free play of this tendon.

He showed photographs of patients treated by this method whose deformities appear to have been corrected without subsequent manipulations or plasters.

The peroneous-longus tenolysis is also employed in certain types of flat feet. He found that in these flat feet, when the heel-valgus was corrected, the patient could not actively depress the 1st metatarsal head onto the ground. These patients therefore had a fixed supinated forefoot due to adhesions of the peroneus longus in its plantar tunnel. When these adhesions were severed the 1st metatarsal head could be actively depressed and the patients became free of deformity and discomfort. He felt that this type of flat foot was not common in the Negro.

In the discussion *Mr. J. M. Edelstein* (Johannesburg) mentioned that in the Bantu foot there is an extra insertion of tibialis anterior in 12%, and the peroneus tertius is often absent. Other anomalies of tendon insertions in the foot occur in 2·5% of Bantu as compared with only 1·5% of Whites, and one would therefore expect a greater evidence of club feet in the Bantu. This deformity, however, does not appear to be more prevalent in the Bantu than in the White population.

Osteotomy of the Os Calcis in the Treatment of Pes Cavus

Mr. F. C. Dwyer (Liverpool) presented a new approach to the treatment of pes cavus. The operation advised by him consists essentially in a subcutaneous division of the contracted plantar fascia and correction of the varus deformity of the heel by removing a wedge of bone from the outer aspect of the os calcis. He submitted that by approaching the deformity from behind and overcoming the varus of the heel, the foot is rendered plantigrade and thereafter weight bearing exerts a corrective influence which results in progressive improvement of the deformity. The operation is essentially a prophylactic one and should be performed before gross structural deformity develops and while active growth is still taking place. He had performed the operation 63 times in 47 children, of ages varying from 3 to 16 years. Even in patients over the age of 16 great improvement is obtained by doing nothing more than this operation. There is improvement in their gait and shoe-wear; and the drop-foot and claw-toe deformities are strikingly corrected. In adults with fixed forefoot deformity the cavus can be corrected at the same time by removing a dorsal wedge from the tarso-metatarsal region, thus preserving movement at the mid-tarsal subastragaloid joint.

He showed photographs and X-rays of patients on whom this operation had been performed, and also a film illustrating the operation.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES

The official list of recognized societies as at 1 November 1956 is published below for general information. This replaces the list in the present Tariff Book.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
10 November 1956

MEDIESE HULPVERENIGINGS

Die offisiële lys van erkende verenigings, met ingang 1 November 1956, word vir algemene inligting hieronder gepubliseer. Dit vervang die lys in die huidige tarieweboek.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
10 November 1956

APPROVED MEDICAL AID SOCIETIES : GOEDGEKEURDE MEDIESE HULPVERENIGINGS

1. A.A. Mutual Medical Aid Society, P.O. Box 9595, Johannesburg.
2. Abercom Group Sick Benefit Society, P.O. Box 715, Port Elizabeth, C.P.
3. African Cables Medical Benefit Fund, P.O. Box 172, Vereeniging, Transvaal.
4. African Explosives Medical Aid Society, P.O. Box 1122, Johannesburg.
5. African Homes Trust Sick Fund, 28 Wale Street, Cape Town.
6. African Oxygen & Acetylene Medical Aid Society, P.O. Box 5404, Johannesburg.
7. Afrikaanse Pers Beperk se Siektefonds, Posbus 845, Johannesburg.
8. Alex. Aiken & Carter Medical Benefit Society, P.O. Box 2636, Johannesburg.
9. Algoa Medical Aid Society, P.O. Box 369, Port Elizabeth, C.P.
10. Anglo-Alpha (Dudfield) Medical Aid Society, Private Bag, P.O. Lichtenburg, Transvaal.
11. Anglo-Alpha (Roodepoort) Benefit Society, P.O. Box 15, Roodepoort, Transvaal.
12. Argus Medical Benefit Society (Cape Argus Branch), St. George's Street, Cape Town.
13. Argus Medical Benefit Society (Daily News Branch), P.O. Box 1491, Durban.
14. Argus Medical Benefit Society (Star Branch), P.O. Box 1014, Johannesburg.
15. A.T.I. Medical Aid Society, P.O. Box 5057, Boksburg North.
16. Atlantic Refining Company Medical Aid Society, P.O. Box 664, Cape Town.
17. Babcock and Wilcox Medical Aid Fund, P.O. Box 545, Vereeniging.
18. Bakers Ltd. European Employees' Sick Benefit Fund, P.O. Box 692, Durban.
19. Bloemfontein Municipal Employees' Medical Aid Society, P.O. Box 288, Bloemfontein.
20. Boksburg Municipal Employees' Medical Aid Fund, P.O. Box 215, Boksburg.
21. Broderick Medical Aid Society, P.O. Box 186, Vereeniging.
22. Building Societies Joint Medical Aid Fund, P.O. Box 5728, Johannesburg.
23. S. Butcher & Sons Ltd. Medical Aid Society, P.O. Box 1004, Durban.
24. Caltex Medical Aid Society (S.A.), P.O. Box 714, Cape Town.
25. Cape Times Medical Aid Society, P.O. Box 11, Cape Town.
26. Cape Town Municipal Employees' Medical Aid Society, P.O. Box 1939, Cape Town.
27. Central News Agency Limited Medical Benefit Society, P.O. Box 1033, Johannesburg (excluding Cape Town and

suburbs, Durban municipal area, Johannesburg and Witwatersrand, and Port Elizabeth and Pretoria municipal areas).

28. Civil Service Medical Benefit Association, P.O. Box 176, Pretoria.
29. Consolidated Glassworks Limited Medical Aid & Sick Benefit Society, P.O. Box 562, Germiston.
30. Corner House Insurance Fund, P.O. Box 1056, Johannesburg.
31. Coronation Medical Aid Society, P.O. Box 1517, Durban.
32. Crookes Bros. Limited Medical Benefit Fund, 301 Smith Street, Durban.
33. D.F.A. Medical Benefit Society, P.O. Box 610, Kimberley.
34. Eastern Province Cement Co. Ltd. Medical Aid Society, P.O. Box 2016, Port Elizabeth.
35. E.P. Newspapers Medical Aid Society, P.O. Box 1117, Port Elizabeth.
36. E.D.C. Industries Medical Aid Society, P.O. Box 3448, Johannesburg.
37. Egnep Medical Aid Society, P.O. Penge, Transvaal.
38. Elwamba Medical Aid Fund, c/o E.L. Wool Exchange Limited, 50/52 Church Street, East London.
39. Escom (Natal Central Undertaking) Medical Benefit Society, P.O. Box 30, Colenso, Natal.
40. Everite Medical Aid Society, P.O. Kliprivier, Transvaal.
41. Federated Employers' Medical Aid Society, P.O. Box 666, Johannesburg.
42. Federation of Master Printers of S.A. Medical Aid Society, P.O. Box 1200, Johannesburg.
43. Ford Medical Aid Society, P.O. Box 788, Port Elizabeth.
44. Friend Medical Aid Fund, P.O. Box 245, Bloemfontein.
45. General Mining (Associated Companies) Medical Aid Society, P.O. Box 1242, Johannesburg.
46. General Motors Medical Aid Scheme, P.O. Box 1137, Port Elizabeth.
47. Germiston Industries Medical Aid Society, 113 Pylon House, Herman Street, Germiston.
48. Gledhow-Chaka's Kraal Sugar Co. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
49. Greaterman's Pension Benefit and Welfare Society (all Branches), P.O. Box 5460, Johannesburg.
50. Hollerith Medical Aid Society, P.O. Box 7018, Johannesburg.
51. Hubert Davis Johannesburg Staff Medical Aid Society, P.O. Box 1386, Johannesburg.
52. Sir J. L. Hulett & Sons Ltd. Medical Benefit Fund, P.O. Box 248, Durban.
53. Hume Cape Medical Benefit Society, P.O. Box 7, Bellville, C.P.
54. Hume (Transvaal) Medical Benefit Society, P.O. Box 204, Germiston.
55. Hunt, Leuchars & Hepburn Ltd. (Durban) Employees' Medical Benefit Fund, P.O. Box 943, Durban.
56. Hunt, Leuchars & Hepburn Ltd. (Transvaal Staff) Medical Aid Society, P.O. Box 47, Johannesburg.
57. Iscor Medical Benefit Fund, P.O. Box 450, Pretoria.
58. I.W.S. Medical Aid Society, P.O. Box 6946, Johannesburg.
59. J. W. Jagger & Co. Ltd. Medical Aid Society, P.O. Box 726, Cape Town.
60. Johannesburg Board of Executors' Medical Aid Society, P.O. Box 271, Johannesburg.
61. Joseph Liddle (Pty.) Ltd. Medical Aid Society, P.O. Box 106, Johannesburg.
62. Klerksdorp Munisipale Werknemers Siektefonds, Posbus 99, Klerksdorp.
63. K. & L. Timbers Ltd., P.O. Box 6994, Johannesburg.
64. Koegas Medical Aid Society, P.O. Koegasbridge, C.P.
65. Krantzberg Mines Medical Aid Society, P.O. Box 18, Omaruru, S.W.A.
66. Kroonstad Munisipale Mediese Hulpvereniging, Posbus 302, Kroonstad.
67. G. H. Langler & Co. Medical Aid Society, P.O. Box 3762, Johannesburg.
68. Legal & General Medical Aid Scheme, P.O. Box 4870, Johannesburg.
69. Mail, Times & Express Medical Aid Society, c/o Rand Daily Mail Ltd., P.O. Box 1138, Johannesburg.
70. L. H. Marthinusen Medical Aid Society, P.O. Box 64, Denver, Johannesburg.
71. Masonite Medical Aid Society, P.O. Box 57, Estcourt, Natal.
72. Metal Box Company of S.A. Medical Aid Society, P.O. Box 7752, Johannesburg.
73. Municipal Employees' Medical Aid Society (Durban), P.O. Box 625, Durban.
74. Natal Building Society Medical Aid Fund, P.O. Box 947, Durban.
75. Natal Coal Owners' (Durban Staff) Medical Aid Society, P.O. Box 281, Durban.
76. Natal Estates Sick Fund Benefit Society, P.O. Mount Edgecombe, Natal.
77. Natal Industries Medical Aid Society, P.O. Box 1300, Durban.
78. N.T.E. Staff Medical Aid Fund, P.O. Box 39, Pietermaritzburg.
79. National Industrial Credit Corporation Medical Aid Society, P.O. Box 8296, Johannesburg.
80. National Portland Medical Aid Society, P.O. Box 21, Claremont, C.P.
81. New Consolidated Goldfields Employees' Medical Aid Fund, P.O. Box 1167, Johannesburg.
82. Northern Medical Aid Society, P.O. Box 3437, Johannesburg.
83. Northern Rhodesia Civil Servants Medical Aid Society, P.O. Box 294, Lusaka, Northern Rhodesia.
84. Norwich Union Life Insurance Society Staff Medical and Surgical Benefit Scheme, P.O. Box 1226, Cape Town.
85. Ore & Metal Medical Aid Society, P.O. Box 3548, Johannesburg.
86. Pietermaritzburg Chamber of Industries Medical Aid Society, P.O. Box 365, Pietermaritzburg.
87. Polliack Group Medical Aid Society, P.O. Box 3008, Johannesburg.
88. Pongola Sugar Milling Co. Ltd. Medical Benefit Fund, P.O. Box 194, Durban.
89. Post Office Medical Aid Society, P.O. Box 303, Germiston.
90. Pretoria Municipal Employees' Sick Fund, P.O. Box 408, Pretoria.
91. Pretoria News Medical Benefit Society, P.O. Box 439, Pretoria.
92. Pretoria Portland Cement Co. Ltd. No. 1 Works (Hercules) Medical Aid Society, P.O. Box 405, Pretoria.
93. Pretoria Portland Cement Co. Ltd. No. 2 Works Medical Society, P.O. Box 7, Slurry, Western Transvaal.
94. Pretoria Portland Cement Co. Ltd. No. 3 Works (Jupiter) Medical Aid Society, P.O. Box 73, Cleveland, Transvaal.
95. Pretoria Portland Cement Co. Ltd. No. 4 Works Medical Aid Society, P.O. Box 26, Orkney, district Klerksdorp.
96. Printing Industry Medical Aid Society, P.O. Box 1993, Pretoria.
97. Rand Public Service Medical Aid Society, P.O. Box 28, Boksburg.
98. Rand Water Board Sick Fund, P.O. Box 1127, Johannesburg.
99. Randles Bros. & Hudson Ltd. (Durban) Sick Benefit Fund, P.O. Box 1046, Durban.
100. Randles Bros. & Hudson Ltd. (Johannesburg) Employees' Sick Benefit Fund, P.O. Box 2678, Johannesburg.
101. Reckitt & Colman Medical Aid Society (S.A.), P.O. Box 1097, Cape Town.
102. 'Rennie' & 'The Consolidated' Employees' Medical Aid Fund, P.O. Box 1006, Durban.
103. Reunert & Lenz Ltd. Medical Aid Society (All Branches), P.O. Box 92, Johannesburg.
104. Reynolds Bros. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
105. E. S. & A. Robinson (Pty.) Ltd. Medical Aid Society, P.O. Box 293, Germiston.
106. Royal-Globe Medical Aid Fund, P.O. Box 317, Cape Town.
107. Safim Medical Aid Society, P.O. Box 233, Vereeniging.
108. Safmarine Medical Aid Society, P.O. Box 2171, Cape Town.
109. Safnit Mills Medical Aid Fund, P.O. Box 11, Jeppestown, Johannesburg.
110. Santam-Sanlam Siektefonds (Alle Takke), Posbus 1, Sanlamhof, K.P.
111. Sasol Medical Aid Society, P.O. Box 1, Sasolburg, O.F.S.
112. Schwartz, Fine, Kane & Co. Medical Aid Society, P.O. Box 5069, Johannesburg.

113. Shell Medical Aid Society (S.A.), P.O. Box 2231, Cape Town.
114. Siektefonds van Wolgroeiers Afslaers Beperk, Posbus 765, Port Elizabeth.
115. C. G. Smith & Co. Ltd. Medical Aid Fund, 301 Smith Street, Durban.
116. S.A. Association of Municipal Employees' (S.A.A.M.E.) Medical Aid Fund, P.O. Box 62, Pretoria.
117. S.A. Breweries Medical Aid Society, P.O. Box 1099, Johannesburg.
118. S.A.K.A.V. Sick Benefit Fund, P.O. Box 33, Paarl.
119. S.A. Mutual Fire & General Insurance Co. Ltd. Staff Medical Aid Fund, P.O. Box 516, Johannesburg.
120. S.A. Mutual Life Assurance Society Staff Medical Aid Fund, P.O. Box 66, Cape Town.
121. S.A. Press Association Medical Aid Society, P.O. Box 7766, Johannesburg.
122. S.A. Teachers' Association Medical Aid Society, 12 Bellevue Road, Sea Point, C.P.
123. S.A. Torbanite (Boksburg) Medical Aid Society, P.O. Box 83, Boksburg North.
124. South Atlantic Corporation Medical Aid Society, P.O. Box 4610, Cape Town.
125. Southern Medical Aid Society, P.O. Box 42, Cape Town.
126. Standard Brass Medical Aid Society, P.O. Box 229, Benoni.
127. Stewarts & Lloyds Medical Benefit Fund, P.O. Box 74, Vereeniging.
128. Stuttafords Medical Aid Society, P.O. Box 69, Cape Town.
129. Sun Insurance Office Ltd. Staff Medical Aid Fund, P.O. Box 429, Johannesburg.
130. Traduna Medical Aid Fund, P.O. Box 8791, Johannesburg.
131. Transvaal Chamber of Mines Medical Aid Society, P.O. Box 809, Johannesburg.
132. Transvaal Corundum Associated Asbestos Medical Aid Society, P.O. Box 72, Pietersburg, Transvaal.
133. Transvaal Society of Accountants Medical Aid Society, P.O. Box 2995, Johannesburg.
134. Umzimkulu Sugar Co. Ltd. Medical Aid Fund, P.O. Box 43, Durban.
135. Union Flour Mills Sick Fund, P.O. Box 1010, Durban.
136. United Banks' Medical Aid Society, P.O. Box 1242, Cape Town.
137. United Building Society Medical Benefit Fund, P.O. Box 7735, Johannesburg.
138. University of the Witwatersrand (Johannesburg) Staff Medical Aid Fund, Milner Park, Johannesburg.
139. Vacuum Medical Aid Society, P.O. Box 35, Cape Town.
140. Village Board of Management of Welkom Medical Aid Society, P.O. Box 708, Welkom, O.F.S.
141. Wright Boag & Head Wrightson Sick Benefit Fund, P.O. Box 183, Benoni.
142. Yorkshire Medical Aid Society, P.O. Box 2755, Johannesburg.

MEDICAL BENEFIT SOCIETIES WHICH ALLOW FREE CHOICE OF DOCTOR FOR SPECIALIST SERVICES ONLY : MEDIESE BYSTANDSVERENIGINGS WAT VRY KEUSE VAN DOKTER ALLEEN VIR SPESIALISTEDIENSTE TOELAAT

1. Begbie Medical Benefit Fund, P.O. Box 192, Middelburg, Transvaal.
2. Breyten Coalfields Benefit Society, P.O. Box 110, Ermelo.
3. Broken Hill Mine Employees' Medical Specialist Fund, P.O. Box 45, Broken Hill.
4. De Beers Consolidated Mines Limited Benefit Society, P.O. Box 616, Kimberley.
5. Durban Roodepoort Deep Limited Benefit Society, P.O. Box 193, Roodepoort.
6. Jagersfontein Mine Benefit Society, P.O. Box 2, Jagersfontein, O.F.S.
7. Krugersdorp Municipal Employees' Medical Benefit Society, P.O. Box 101, Krugersdorp.
8. Northern Rhodesia Mine Employees. Medical Fund, P.O. Box 134, Kitwe, Northern Rhodesia.
9. Public Utility Transport Corporation Staff Sick Fund, P.O. Box 9571, Johannesburg.
10. Randfontein Estates Employees' Sick Benefit Society, P.O. Box 37, Randfontein.
11. Roodepoort-Maraisburg Municipal Employees' Association Sick Benefit Society, P.O. Box 217, Roodepoort.
12. Roodepoort-Maraisburg Non-Scheduled Mines' and Industries' Benefit Society, P.O. Box 225, Roodepoort.
13. Rosherville-Maraisburg Benefit Society, P.O. Box 99, Cleveland, Johannesburg.
14. Simmer Pan Medical Benefit Society, P.O. Box 103, Germiston.
15. Springs Mines Benefit Society, P.O. Box 54, Springs.
16. Springs Municipal Employees' Association, P.O. Box 45, Springs.
17. Transvaal Jewellers' and Goldsmiths' Sick Benefit Fund, P.O. Box 8530, Johannesburg.
18. Vereeniging Power Station Medical Society, P.O. Box 27, Vereeniging.
19. Witbank Coalfields Benefit Society, P.O. Box 26, Witbank.
20. Witbank Power Station Medical Aid Society, P.O. Box 197, Witbank.

Note (1) The Federal Council of the Medical Association of S.A. has extended the operation of the Tariff to the personnel and dependants, pensioners and their dependants of the High Commission Territories (Basutoland, Bechuanaland Protectorate and Swaziland). Persons entitled to such concessions will be supplied with letters of introduction signed by the Secretaries of the Territories concerned.

Note (2) The Tariff also applies to additional medical assistance rendered to members of the Police Force, Prison Officers, members of the Permanent Defence Forces and the wives and unmarried children under the age of 18 years of European personnel of these Departments. In addition it applies to all persons for whose medical attention the Government is responsible in the Departments of Education, Arts and Science; Health; Pensions and Social Welfare. Such fees will be subject to a reduction of 10% on services to a patient costing up to 25 guineas and of 15% on those costing over 25 guineas, but no reduction will apply to any account of £2 2s. 0d. or less in respect of services rendered to any one patient.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. D. J. Retief who has recently returned from England and America after 4 years postgraduate work in orthopaedic surgery has joined the practice of Mr. G. T. du Toit, 73 de Villiers Street, Johannesburg.

* * *

Dr. D. J. Retief, wat onlangs van Engeland en Amerika teruggekeer het na 4 jaar na-graadse studie in ortopiese snykunde, het toegetree tot die praktyk van Dr. G. T. du Toit, de Villiersstraat 73, Johannesburg.

Mr. W. Silber, M.B., Ch.B. (Cape Town), F.R.C.S. (Edin.), formerly Registrar, Hammersmith Hospital (Postgraduate Medical School), London, has commenced practice in Cape Town as a specialist surgeon. Telephones 7-4458 and 7-7727.

* * *

Physical Medicine Group. At an International Congress recently held at Copenhagen the South African Society of Specialists in Physical Medicine was elected a full Member of the International Federation of Physical Medicine.

Union Department of Health Bulletins. Report for the 7 days ended 8 November 1956.

Plague, Smallpox: Nil.

Typhus Fever: One (1) Native case in Uitenhage district and one (1) Native case in Umtata district. Diagnosis based on clinical grounds only. One (1) Native case in Queenstown district and one (1) Native case in Queenstown Native Location. Diagnosis confirmed by laboratory tests as positive.

Epidemic Diseases in Other Countries.

plague: Nil.

Cholera in Calcutta (India); Chalna, Chittagong, Dacca (Pakistan).

Smallpox in Rangoon (Burma); Allahabad, Bombay, Calcutta, Cuddalore, Jodhpur, Karikal, Madras, Pondicherry, Tiruchirappalli, Tuticorin (India); Makassar (Indonesia); Mosul (Iraq); Dacca (Pakistan).

Typhus Fever: Nil.

Report for the 7 days ended 15 November 1956.

Plague, Smallpox: Nil.

Typhus Fever: (One (1) Native case in the Umtata district, reported in Bulletin No. 45 has now been confirmed by laboratory tests as positive. One (1) Native Case in the Cradock Native location, one (1) Native case in the Queenstown district and one (1) Native case in the Fort Beaufort district. These three cases have been confirmed by laboratory tests as positive. One (1) Native case in the Uitenhage district reported in Bulletin No. 45 has now been diagnosed as Tick Bite Fever.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Calcutta (India); Chalna (Pakistan).

Smallpox in Kandahar (Afghanistan); Calcutta, Karikal, Lucknow, Tiruchirappalli, Visakhapatnam (India); Baghdad, Mosul (Iraq).

Typhus Fever in Mombasa (Kenya).

* * *

Dr. F. W. Quass, D.Sc., has been appointed by the South African Council for Scientific and Industrial Research as Director of the National Nutrition Research Institute. He is at present technical adviser to the Natural Resources Development Council.

South African Paediatric Association. The next meeting of the Cape Town Sub-group of the Society will be held on Friday 7 December 1956 in the E-Floor Lecture Theatre, Groote Schuur Hospital, Cape Town, at 8.15 p.m. The subject for the evening will be 'The Problem of the Hard-of-Hearing Child', which will be introduced by Dr. N. van der Merwe, of the Department of Education. Miss W. Thomas, of the Hard-of-Hearing Centre, Mowbray, will talk on the rehabilitation of the hard-of-hearing child and Dr. D. Maytham will discuss the diagnosis and treatment of deafness in childhood.

* * *

Royal College of Physicians of Edinburgh. At a meeting of the College held on 6 November, the President, Sir Stanley Davidson, in the chair, Albert Rabinowitz, M.D. (Rand), was elected a Fellow of the College. The following also were elected Members of the College: Reuben Sougin-Mibashan, M.D. (Cape Town); Amir Khan, B.A., B.Sc., M.B. (Rand); Francis Chemfaa Wong Lunking, M.B. (Rand); Denis Michael Krikler, M.B. (Cape Town) and Leon Charles Isaacson, B.Sc., M.B. (Cape Town).

* * *

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria.

Failure to advise the Association can only result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as to those who change their addresses within the Union.

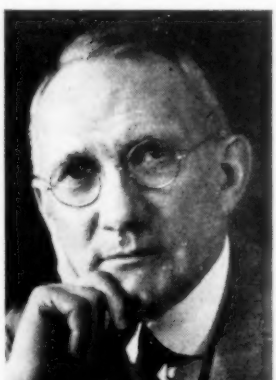
* * *

Dr. Theodore Leonard Sarkin, M.D., Ch.M. (Cape Town), who has been working in the United Kingdom on a Cecil John Adams Travelling Fellowship, has obtained the F.R.C.S. both in Edinburgh and London. Dr. Sarkin intends visiting the United States, and will return to Groote Schuur Hospital, Cape Town, from which he is at present on leave, in March 1957.

IN MEMORIAM

R. C. J. MEYER, M.D. (EDIN.), F.R.C.S. (EDIN.)

Dr. Maurice Franks writes: Dr. R. C. J. Meyer died at the age of 73 in Johannesburg on 7 November 1956.



Dr. R. C. J. Meyer
[Photo Levson, Johannesburg

Educated at Cape Town he took a B.A. degree at the University and won a Jamieson scholarship, after which he proceeded to Edinburgh, where he graduated M.B., Ch.B. in 1908. He returned to South Africa in 1910 and remained here until 1912, after which he returned to Edinburgh to take his M.D., F.R.C.S., as well as the D.O. (Oxford).

When the war broke out, he joined the Royal Navy and saw service from 1914 to 1918 in various parts of the world.

On return to civilian life, Dr. Meyer became house surgeon at Moorfield's, London, and then returned to South Africa to set up private practice in Johannesburg. He was appointed Ophthalmic Surgeon to the Transvaal Memorial Hospital for Children in 1923 and to the Johannesburg General Hospital in 1924, where he served until his retirement in 1938. At my request he again resumed duty at the General Hospital during 1940-1945 to take the place of ophthalmic surgeons who were on active service.

During the past two years his health has not been of the best; nevertheless he continued in practice and, in fact, was in consultation on the morning before his death.

In 1930, together with the late Dr. D. J. Wood, he was instrumental in founding the Ophthalmological Society of South Africa, of which, in 1931, he was secretary of the Johannesburg section. In 1938 and 1939, he was President of the O.S.S.A. Early this year he was awarded the Emeritis Membership of the O.S.S.A. in recognition of his services to the Society, this being the first occasion of such an award.

'Reinkie' was a most punctilious surgeon. He could never be hurried and did all his work with extreme care and consideration. His ability as a surgeon was recognized throughout South Africa, and his understanding of his patients endeared him to all who knew him or benefited from his ministration. He was a forthright person and did not hesitate to express his opinion of approval or disapproval. He hated any kind of sham, whether medical or social.

His passing has left a gap in the ranks of the ophthalmologists of South Africa, who benefited so much from his advice and counsel. To his widow and children, 3 of whom are practising as members of our profession (one in ophthalmology), we extend our deepest sympathy on the passing of one who earned the respect and love of all with whom he came in contact.

REVIEWS OF BOOKS : BOEKRESENSIES

ANAESTHETICS

A Manual of Anaesthetic Techniques. By William J. Pryor, M.B., Ch.B. (N.Z.), F.F.A.R.C.S. (Eng.), D.A. (Eng.), M.F.A., R.A.C.S. and a foreword by J. H. T. Challis, M.R.C.S. (Eng.), L.R.C.P. (Lond.), F.F.A.R.C.S. (Eng.), D.A. (Eng.). Pp. viii + 224 + 77 illustrations. 27s. 6d. Bristol: John Wright & Sons, Ltd. 1956.

Contents: I. The Anaesthetic Room. II. Equipment. III. Mechanical and Physiological Aspects of Anaesthesia. IV. Agents used in Anaesthesia. V. Pre-operative Conditions requiring Special Care in Anaesthesia. VI. Intubation. VII. Premedication and Basal Narcosis. VIII. General Anaesthetic Techniques. IX. Machines used for Controlled Respiration. X. Special Anaesthetic Techniques. XI. Anaesthesia in Children. XII. Geriatric Anaesthesia. XIII. Dental Anaesthesia. XIV. Emergency Anaesthesia and Resuscitation. XV. Anaesthetic Emergencies and Difficulties. XVI. Post-operative Care: Complications and Treatment. XVII. Spinal Analgesia. XVIII. Local Analgesia. XIX. Intravenous Therapy. XX. Hypotension. XXI. Antihistamines in Anaesthesia. XXII. The Explosion Risk. XXIII. The Expanding Role of the Anaesthetist. Index.

The author in an easy direct and comprehensible style has succeeded in producing a most valuable book on the practical aspects of Anaesthesia. It is a book primarily written for the beginner but should prove of great value to those concerned with the teaching of Anaesthesia and also as a book of ready reference to the busy anaesthetist. All the fundamentals of all aspects of anaesthesia are more than adequately handled.

As stated in his preface to this work, the author has wisely given the anaesthetist a choice of well-tried methods in the section dealing with technique, placing emphasis on the ability of the operator to employ any given method rather than the technique itself.

The whole work is concise, yet nothing of importance has been omitted and this book should provide a healthy stimulus to wider reading on those special aspects of Anaesthesia which are the province of the specialist anaesthetist.

L.M.v.d.S.

ENDOCRINOLOGY

Year Book of Endocrinology—1955-1956 *Series.* Edited by Gilbert S. Gordan, M.D., Ph.D. Pp. 367. 82 Figures. $6.00. Chicago: The Year Book Publishers, Inc. 1956.

Contents: Introduction. *The Pituitary Glands.* Adenohypophysis: Adenohypophysis and Diabetes Mellitus, Hypopituitarism, Growth Hormone and Acromegaly, Pituitary Tumors. Neurohypophysis and Water Metabolism: Familial Diabetes Insipidus. *The Thyroid Gland.* Tests of Thyroid Functions. Transport of Hormonal Iodine. Triiodothyronine and Triiodothyroacetin Acid. Endemic Goitrogens. Hyperplastic Hypothyroidism. Hypothyroidism. Myxedema Coma. Hyperthyroidism. Exophthalmos. Thyroid Cancer and Thyroiditis. *The Parathyroid Glands, Calcium Metabolism and Metabolic Bone Diseases.* Parathyroid Function Tests. Hyperparathyroidism. Pseudohyperparathyroidism. Hypoparathyroidism. Pseudohypoparathyroidism and Pseudopseudohypoparathyroidism. Osteomalacia, Osteoporosis and Albright's Syndrome. *The Adrenal Glands.* Adrenal Medulla. Adrenal Cortex: Adrenal Cortical Hormones, Aldosterone, Primary Aldosterone and Adrenal Tumor, by E. Perry McCullagh, Tests of Adrenal Cortical Functions, 'Stress' and Adrenal Corticoids, Addison's Disease, Adrenal Virilism, Cushing's Disease and Cushing's Syndrome. *The Reproductive System.* Gonadal Dysgenesis, Hermaphrodism and Pseudohermaphrodism. Pregnancy and Gonadotrophins. Ovarian Virilization. Estrogens. Hormonal Influences on Lipids and Atherosclerosis. Male Hypogonadism. Androgens. *Carbohydrate Metabolism.* Hypoglycemic Action of Sulfonamide Derivatives in Diabetes Mellitus, by L. Arthur Mirsky. Insulin and Insulinase Inhibitors. Insulin Preparations. Hyperinsulinism and Hypoglycemia. Glucagon. Diabetes Mellitus. Complications of Diabetes Mellitus. *Endocrine Treatment of Neoplastic Diseases.* Carcinoma of the Breast. Carcinoma of the Prostate.

This very welcome annual has reappeared with its usual excellent review of the year's most significant papers in the field of endocrinology. It is remarkable that so much material has been compressed into a volume containing less than 350 pages. Yet the abstracts are both accurate and easily readable. Credit must go to the editor, Dr. Gordan, whose pithy and pertinent comments reveal his expert understanding of this expanding subject. The volume is quite up-to-date, reference being made to articles published quite recently.

The annual considers each endocrine gland individually and covers all important advances in physiological, experimental and clinical aspects. In addition there are chapters devoted to the role of hormones in breast and prostatic carcinoma; to the blood lipids and atherosclerosis; and to non-endocrine metabolic bone-disorders. Apart from the abstracts there are two special articles of importance. The first is by McCullagh, who deals with aldosterone and adrenal tumours. The realization of this hormone's significance rates as one of the major advances of recent years; the full implications have yet to be appreciated. In another article Mirsky discusses the available literature on the new insulin-sparing sulphonamide derivatives. While it is clear that these have considerable limitations, they do hold some promise of future oral therapy in diabetes mellitus. This prompt report will be welcomed by all who have to deal with diabetic patients.

Today no physician—or general practitioner—can afford to ignore the endocrine field. There can be no better or simpler means of keeping abreast than by perusing this volume. It is very strongly recommended to all readers.

R.H.

NEUROLOGY AND PSYCHIATRY IN CHILDHOOD

Neurology and Psychiatry in Childhood. Volume XXXIV. Proceedings of the Association December 10 and 11, 1954. New York, N.Y. Pp. xii + 504. 63 Illustrations and 21 Tables. 88s. London: Baillière, Tindall & Cox, Ltd. 1954.

Contents: Part I. Infections of the Central Nervous System. I. Treatment o Puogenic Meningitis. Hattie E. Alexander. II. Tuberculous Meningitis. Ralph V. Platou and John H. Arnold. III. Present Status of the Problem of Vaccination Against Poliomyelitis. Jonas E. Salk. IV. Encephalitides and Postinfectious Encephalopathies. Horace L. Hodes. *Part II. Developmental and Traumatic Aspects.* V. Intracranial Hemorrhage in Infancy and Childhood. Donald D. Matson. VI. Developmental Defects of the Skull and Spine Associated with Lesions of the Central Nervous System. Edgar A. Kahn. VII. Injury of the Central Nervous System Incurred During Fetal Life. Samuel P. Hicks. VIII. The Formation, Flow, and Absorption of Cerebrospinal Fluid: Newer Concepts Based on Studies with Isotopes. William H. Sweet, Gordon L. Brownell, John A. Scholl, David R. Bowsher, Philippe Benda and E. F. Stickley. IX. Hydrocephalus. Dorothy S. Russell. X. Hemispherectomy in the Trea ment of Convulsive Seizures Associated with Infantile Hemiplegia. Joseph Ransohoff and Sidney Carter. *Part III. Functional and Degenerative Disturbances.* XI. The Cerebral Atrophies and Encephalomalacias of Infancy and Childhood. Abner Wolf and David Cowen. XII. Myasthenia Gravis in Infants and Children. Rhett P. Walker. *Part IV. Roentgenographic Aspects.* XIII. Some Radiological Features of Traumatic Lesions in the Growing Skull. John Caffey. XIV. The Contour of the Skull in the Presence of Increased or Diminished Intracranial Pressure. Edward B. D. Neuhauser. *Part V. Psychiatric Aspects.* XV. A Research Approach to Reading Retardation. Ralph D. Rabinovitch, Arthur L. Drew, Russell N. De Jong, Winifred Ingram and Lois Withey. XVI. The Epidemiology of Behaviour Disorders of Childhood. Benjamin Pasamanick. XVII. The Psychiatric Approach to Posttraumatic and Postencephalitic Syndromes. Abram Blau. XVIII. Affective Deprivation and Early Institutional Placement. J. Franklin Robinson. XIX. The Role of the Clinical Psychologist in the Evaluation of Children with Specific Handicaps. Samuel J. Beck. *Part VI.* Symposium on Juvenile Schizophrenia. XX. General Concept of Schizophrenia at Different Ages. Leo Kanner. XXI. Problems in the Differential Diagnosis of Childhood Schizophrenia. J. Cotter Hirschberg and Keith N. Bryant. XXII. Treatment of Juvenile Schizophrenia. Lauretta Bender. List of Members. Index.

This volume contains the proceedings (papers and discussions) of the 34th Annual Meeting of the Association of Research in Nervous and Mental Disease, held in 1954. At the request of the editor certain authors have submitted more detailed accounts for publication than were actually presented at the meeting.

The arrangement of a programme for such a meeting is understandably a most difficult task. The omission of the convulsive disorders in childhood will immediately strike the reader. The editors themselves mention this in their preface.

Discussing the bacterial meningitides (exclusive of tuberculous meningitis) the author calls serious attention to the common occurrence of subdural collections of fluid in early life. It is her policy to explore the subdural space in all infants with open sutures the day before treatment is discontinued, unless the signs indicate the presence of fluid earlier. She is unwilling to make burr-holes as a routine in children older than 1 year.

The authors of the paper on tuberculous meningitis advocate prophylaxis of this disease by treatment of the primary complex in the first 2 years of life by isoniazid alone. They state that no one in the US has reported the development of tuberculous meningitis whilst such cases were being treated with isoniazid only.

Salk discusses the status (in December 1954) of vaccination against poliomyelitis. At that time he could state 'prophylaxis with live virus was only an idea', and 1954 in this connection seems a very long time ago. Salk and Koprowski and Sabin have taken that idea a good way into practical experience since then, and attenuated poliovirus by mouth for prophylaxis is past the stage of an idea.

The paper on Encephalitides and Post-infectious Encephalopathies is by Hodes.

Part II deals with intracranial haemorrhage in infancy and childhood, injuries of the CNS incurred during foetal life, and developmental defects of skull and spine. The contribution by Dorothy Russell on Hydrocephalus is the substance of her MRC paper on the subject. The place of hemispherectomy in convulsive seizures associated with infantile hemiplegia is dealt with. This section also contains the recent work on the formation, flow and absorption of the CSF studied with isotopes. The CSF is not fully and completely formed in any one part of the brain—the process is much more complicated.

Part III: The paper on the cerebral atrophies and encephalopathies covers pp. 199-324 and is practically a monograph. The authors stress the importance of cerebral anoxia in the causation of these pathological states. The variability in the intensity and distribution of the lesions may depend on the sensitivity of the given brain to hypoxia and the known variability of resistance of different portions of the brain to oxygen lack, or the metabolic requirements of the brain tissue at the time of exposure. The authors believe that not only may convulsions result from brain damage but that they may actually cause permanent brain-injury in the form of ischaemic necrosis.

Myasthenia gravis in infants and children is dealt with in 4 pages.

In Part IV Caffey discusses with a number of good plates, the radiological features of traumatic lesions of the growing skull. Dr. Neuhauser writes on roentgen effects of altered intracranial pressure. His subject includes craniostenosis.

Part V deals with psychiatric problems:

(*a*) Reading disability, due either to brain damage or to personality factors.

(*b*) Behaviour disorders. The author says: 'It has been shown fairly conclusively that delinquency in children between 10 and 17 in the vast majority of cases is not of psychogenic or psychiatric origin but is rather a sociologic phenomenon'. He hands over the problem to the sociologist for preventive measures. He and his co-workers have studied the question of brain injury at birth as the cause of behaviour disorder. They incline to the view that these injuries are due not so much to prolonged labour but to non-mechanical causes such as toxaemias and hypertensive disease of pregnancy.

(*c*) The author of the next paper, on the psychiatric approach to post-traumatic and post-encephalitic syndromes, says: 'A pure organic behaviour disorder without significant psychopathology is now found extremely rarely. Psychodynamic factors prior to the infection or accident are common. The organic illness often has injurious emotional effects and many environmental, familial and other external conditions continue to disturb the child after the injury'. He had himself written on that basis of these behaviour disturbances in the past. He thinks today that they are mostly anxiety neuroses.

(*d*) Affective deprivation and early institutional placement. Usually under 3 especially under 1, and possibly up to 5 or 6, the infant reacts first with indications of distress or protest and later with withdrawal and unresponsiveness. A personality syndrome has been observed and described by a number of authors.

(*e*) Psychologic evaluation of handicapped children.

Part VI is a symposium on juvenile schizophrenia—a difficult subject.

F.F.

DEPRIVED CHILDREN

Deprived Children. By Hilda Lewis, M.D. (Pp. 163 + xvii. 9s. 6d.) London: Oxford University Press. 1954.

Contents: 1. The Reception Centre and Its Work. 2. The Children: Mode of Admission, Family Background, and Previous Personal Experiences. 3. The Children: Personality and Patterns of Behaviour. 4. Influence. 5. Subsequent Histories: Outcome. 6. General Reflections. Appendix 1. Appendix 2. Index.

Between October 1947 and July 1950, 500 children living in Kent were taken into Mersham Reception Centre, which had been set up by a grant from the Nuffield Foundation. The children were referred to the Centre because they were believed to need care away from their own homes; some had never had a home or had been deprived of it when very young. The Centre, besides caring for the children, gave opportunities for studies which form the subject-matter of this book.

Written by Dr. Hilda Lewis, the psychiatrist to the Centre, the book describes 'the background of the children, the homes they came from, their behaviour, their personalities, the measures taken to help them, and the outcome during the next two years'. When the author writes about background, homes and measures taken, she is on reasonably firm ground, but difficulties arise in the approach to behaviour and personality problems. Our methods of measuring the effect of environment on human behaviour are still crude and lack rigid scientific standards. There is the difficulty, too, referred to by Sir Arthur Ellis and Dr. C. P. Blacker in the foreword, that 'associations between background history and presenting symptoms may in part be genetically determined, for parents may transmit to their children some of the characteristics which brought difficulties and stresses upon themselves.'

In spite of these criticisms, there is a great deal of valuable material in this study, which should be read by everyone interested in child welfare. One would like to see this book, concerned with so human a problem, condensed and published in simpler form, i.e. in a style which avoids the use of psychologic jargon, figures, and statistical tables.

I.M.

SURGERY OF THE SYMPATHETIC

Surgery of the Sympathetic. By Alexander Lee McGregor, F.R.C.S. (Eng.). M.Ch. (Edin.). Pp. 192, with illustrations. 30s. Bristol: John Wright & Sons Ltd. 1955.

Contents: 1. Introduction. 2. The Gross Anatomy of the Autonomic Nervous System. 3. The parasympathetic. 4. Surgical Anatomy and Physiology. 5. Sympathetic Arrangements in the Head and Neck. 6. Cerebral Thrombosis and Embolism. 7. Sympathetic Arrangements in the Upper Limb. 8. Sympathetic Denervation of the Upper Limb. 9. Sympathetic Arrangements in the Lower Limb. 10. Sympathetic Denervation of the Lower Limb. 11. The Prognosis following Sympathetic Denervation of the Limbs. 12. The Clinical Presentation of Sympathetic Derangements. 13. The Heart. 14. The Lung. 15. The Abdominal Sympathetic System. 16. Abdominal Pain. 17. Neuromuscular Inco-ordination. 18. The autonomic Arrangements in the Pelvis. 19. The Urinary Bladder. 20. The Internal Female Genitalia. 21. Essential Hypertension. 22. The Operations for High Blood-pressure. 23. Hypertension due to Tumours of Chromaffin Tissue. References. Index.

This is essentially a book for the practising surgeon, who will find many valuable suggestions and much technical detail in this short treatise.

The author makes use of and enlarges on the simple diagrams for which he is so well known through his Synopsis of Surgical Anatomy. In some instances, however, simplification is driven too far, and then the diagram may become meaningless. An example of this is the illustration depicting the internuncial pool of Lorento de No, which could with advantage be replaced by one of Lorento de No's original diagrams.

Some of the information contained in the book is inaccurate and therefore misleading, particularly to those not dealing with this discipline every day and turning to the book for guidance. For example, concerning intermittent claudication, the author says he has been informed that this is cured by replacing the diseased main vessel by blood-vessel grafts; and the chapter on essential hypertension, in which the operative technique is admirably described, is, as a whole, out of date.

Nevertheless the practising surgeon will find much of value in this book.

R.H.G.

SIRKULASIESPOED BY KINDERS MET AANGEBORE HARTGEBREKE

Circulatietijden Bij Kinderen Met Aangeboren Hartgebreken. Bepaald Met Behulp van Kleurstofinjectie. By D. H. Beekhuis. Pp. 121, with 29 illustrations. Geb. f. 11.00. Assen: Van Gorcum & Comp. N.V. 1955.

Contents: Circulatietijden Bij Normale Kinderen. 2. Circulatietijden Bij Kinderen met Een Shunt van Rechts naar Links. 3. Circulatietijden Bij Kinderen Met Een Shunt van Links Naar Rechts. 4. Circulatietijden Bij Kinderen Met Een Gemengde Shunt.

Na 'n oorsig oor vorige metodes van sirkulasie-spoedbepaling beskryf die outeur, na bespreking van eksperimentele proewe en vorige werk op die gebied, 'n wyse van vasstelling van die sirkulasiespoed by normale kinders en dié met aangebore hartgebreke; 'n baie waardevolle waarneming, mits dit skadeloos is en betroubaar blyk te wees.

Hy gebruik ‚Evans blue' T. 1824 as 'n enkel inspuiting voldoende is, en metileen blou, waar herhaling nodig is.

'n Brinkman refleksieoksimeter word gebruik om die kleurveranderinge in die vel (afhanklik van die kleurveranderinge in die deurstromende bloed) te meet, en die veranderinge word op 'n grafiek vasgelê, waarvan die verskillende tye bereken word.

Deur die kleurstof perifêr of/en deur 'n hartkateter in verskillende hartkamers in te spuit, word verskillende kurwevorms gekry wat die hart-of vatgebrek aandui. Sy werk is 'n belangrike bydrae tot die studie van hierdie gevalle.

J.G.A.D.

STEAD'S ELEMENTARY PHYSICS

Elementary Physics. By G. Stead, M.A. (Cantab.), D.Sc. (Lond.), F.Inst.P. Ninth Edition. Pp. 532, with 448 illustrations. London: J. & A. Churchill Ltd. 1955.

Contents: Section I. Mechanics. Section II. Hydrostatics and Properties of Matter. Section III. Heat. Section IV. Sound. Section V. Light. Section VI. Magnetism. Section VII. Electrostatics. Section VIII. Current Electricity.

There must be many thousands of practising doctors who have reason to be grateful to the author of this book for the help they received from it during their student days. It has gone steadily on since 1924 through 8 editions and 3 reprintings, and this 9th edition merely brings an excellent text-book up to date.

The last two editions have included a brief sketch of nuclear physics and a reference to radio-active isotopes. There is no doubt that Stead's 'Elementary Physics' will continue to play its part in medical education.

A.H.T.

TOXAEMIAS OF PREGNANCY

The Pregnancy Toxaemias or the Encymonic Atelositeses. By G. W. Theobald, M.A., M.D. (Cambridge), F.R.C.S. (Edin.), F.I.C.S., F.R.C.O.G., M.R.C.P. (Lond.). Pp. 488 + xiv, with 56 illustrations. 63s. 0d. London: Henry Kimpton. 1955.

Contents: 1. Le Milieu Intérieur. 2. The Pregnancy-Lactation Syndrome. 3. The Capillaries. 4. The Water Metabolism of the Body. 5. The Kidneys: Physiological Considerations. 6. The Kidneys: Physiological Proteinuria. 7. Oedema. 8. The Post-Pituitary Gland and Pregnancy. 9. The Adrenal Cortex. 10. Water Diuresis During Pregnancy. 11. The Liver. 12. The Blood Pressure: Physiological Considerations. 13. Hypertension: Essential Hypertension in the Non-Pregnant State. 14. Hypertension During Pregnancy. 15. Hypertension; and Hypertension Associated with Proteinuria. 16. The Toxaemias of Early Pregnancy. 17. Toxaemic Accidental Haemorrhage. 18. Eclampsia. 19. Eclampsia (continued). 20. Eclampsia (Clinical Investigations). 21. The Histological Changes Associated with Eclampsia. 22. Calcium, Magnesium, Phosphorus, Potassium, and Sodium. 23. 'Toxaemias' Associated with Pregnancy in Domestic Animals. 24. Summary of the Phenomena Associated with Pregnancy. 25. The Eclamptic Hypotheses. 26. Placental Hypotheses. 27. Placental Hypotheses (concluded). 28. Placental Hypotheses (continued). 29. The Endocrine Glands. 30. The Crush Syndrome and Renal Ischaemia. 31. The Nutritional-Mechanical Hypothesis. 32. The Nutritional-Mechanical Hypothesis (continued). 33. The Nutritional-Mechanical Hypothesis (continued). 34. The Nutritional-Mechanical Hypothesis (concluded). 35. Ante-Natal Care. 36. The Treatment of 'Pre-Eclampsia'. 37. The Treatment of Eclampsia. 38. The Treatment of Eclampsia (continued). 39. The Treatment of Eclampsia (concluded). Index.

A truly 'British' book on the pregnancy toxaemias has been long overdue. Theobald has now filled this breach with 'The Pregnancy Toxaemias or the Encymonic Atelositeses'. Every known aspect of the pregnancy toxaemias and eclampsia has been covered in great detail.

The book is divided into 4 sections—one following the other in natural sequence, yet each covering an intensive subject on its own. In *Part I* the author deals with the normal metabolic changes found in pregnancy, i.e. in 'the pregnancy—lactation syndrome'. Gradually the author works up to the hypertensive states. In *Part II* the toxaemic states are described, commencing with emesis and hyperemesis gravidarum, covering sea-sickness, heartburn and the other illnesses which may be associated with, or confused with, toxaemias. Accidental haemorrhage and eclampsia are thoroughly done. Theobald then turns to the basic metals and pregnancy toxaemias in domestic animals. This section of his book ends with a 'summary of the phenomena associated with normal and abnormal pregnancy'.

Part III is confined to hypotheses—after the author has first laid down the eclamptic criteria, and *Part IV* to 'The Prevention and Treatment of Eclampsia'.

This book is well written and beautifully balanced. It should be on the shelf of every doctor interested in obstetrics and should be thoroughly studied by all those who are actively engaged in obstetrics—more so by teachers of the subject.

J.T.L.

CORRESPONDENCE : BRIEWERUBRIEK

CARDIAC ARREST FROM TRACTION REFLEX

To the Editor: Dr. Kihn's case report[1] in the *Journal* of 13 October makes very interesting reading. Gallamine triethiodide, being a vagal ganglia inhibitor, should theoretically exert a certain degree of protection against the traction reflex. It is interesting to speculate whether, had atropine, perhaps with ephedrine, been injected intravenously at the time the systolic pressure dropped to 75 mm. Hg for a few minutes, the progressive deterioration would have been averted. Methylamphetamine hydrochloride can cause a bradycardia, *via* vagal effect, and is not an agent recommended. The vagal escape mechanism in cases similar to the one described acts by impulses starting in the conduction tissue below the sino-atrial node. In a diseased myocardium this mechanism is unlikely. The pre-operative ECG findings, if available, would be of interest. It might have been advisable, after the first cardiac arrest, to inject the vagus at the oesophageal hiatus with 1% procaine before proceeding further. This is a useful manoeuvre in any intra-abdominal procedure, particularly in the upper abdomen, where traction is almost unavoidable.

S. G. Paletz

Department of Anaesthesia
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Canada

11 November 1956

1. Kihn, R. B. (1956): S. Afr. Med. J., **30**, 977.

DIE VOORGESTELDE BEHEERREGULASIES VIR BLOEDOORTAPPINGSDIENSTE

Aan die Redakteur: Graag wil ek u felisiteer met die kalme beredenering wat u aan die dag lê in u inleidingsartikel[1] van 27 Oktober 1956, oor die voorgestelde beheerregulasies vir bloedoortappingsdienste. Sonder enige emosionele vertoon bespreek u die handeling en sien dit in die regte lig.

Dit in teenstelling met 'n inleidingsartikel in 'n ander mediese tydskrif in ons land, welke inleidingsartikel beter in die leke-pers tuis hoort.

Dit is betreurenswaardig dat politieke neigings in ons land selfs vir die mediese wetenskap soms oogklappe aansit. As medici kan ons nie afsydig staan teenoor ons pasiënte se emosionele gevoelens en sentimente nie. Dit is egter ons plig om daardie elemente, wat spanning verwek, sover moontlik te matig. Dit is feit dat in ons land daar by 'n meerderheidsgroep van blankes 'n vooroordeel teen nie-blankes bestaan.

Noudat bloed-apartheid in die leke-pers sowel as in die mediese pers groot prominensie verkry het, is daar alreeds 'n groot element van vrees by baie persone dat indien hulle bloed nodig sou kry, nie-blanke bloed in hulle are ingespuit sal word. Hierdie element van spanning is geensins ten voordele van ons pasiënte nie. Ongelukkig het die feite dit dat die grootste bloed-apartheidspropaganda, in die mediese, sowel as leke-pers, deur medici aangevuur is. Sekerlik is daardeur geen guns aan die publiek bewys nie.

Gedane sake het geen keer, maar laat ons deur kalmte van optrede die vertroue van die publiek herwin, sodat hulle sonder vrees en sp nning kan weet dat selfs in noodgevalle hulle geneesheer slegs d e beste ten bevordering van hulle welsyn sal doen.

Aangesien tyd 'n goeie geneesmiddel is, sal dit goed wees indien hierdie hele saak so gou moontlik aan oorbodige perspublisiteit onttrek word.

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